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CATARACT OF DYSTROPHIA MYOTONICA

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After Erb's classic description of Thomsen's disease in 1886, several so-called atypical cases (Thomsen's disease with muscular atrophy) were described sporadically. Deleage reported the first of these in 1890. But it remained for Batten and Gibb [1] and Steinert [2] in 1909 to correlate these reports with cases of their own and to describe myotonia atrophica, a disease entity distinct from other neuromuscular maladies. The chief interest in the syndrome centered about the myotonia and atrophy, with the extramuscular symptoms being considered incidental. However, after Greenfield,[3] Kennedy and Oberndorf [4] and Ormond [5] had described cataracts in some of their cases, Curschmann [6] recognized the significance of the extramuscular manifestations and proposed the name dystrophia myotonica for the disease. Subsequent reports, made principally in the European literature,[7] have brought the total number of cases to approximately 300.

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1. Batten, F. E., and Gibb, H. P.: Myotonia Atrophica, Brain **32**:187-205, 1909.

2. Steinert, H.: Ueber das klinische und anatomische Bild des Muskelschwundes der Myotoniker, Deutsche Ztschr. f. Nervenh. **37**:38-104, 1909.

3. Greenfield, J. G.: Notes on a Family of "Myotonia Atrophica" and Early Cataract, with a Report of an Additional Case of "Myotonia Atrophica," Rev. Neurol. & Psychiat. **9**:169-181, 1911.

4. Kennedy, F., and Oberndorf, C. P.: Myotonia Atrophica, J. A. M. A. **57**: 1117-1118 (Sept. 30) 1911.

5. Ormond, A. W.: A Case of Myotonia Atrophica with Cataracts, Tr. Ophth. Soc. U. Kingdom **31**:214-217, 1911.

6. Curschmann, H.: Ueber familiäre atrophische Myotonie, Deutsche Ztschr. f. Nervenh. **45**:161-202, 1912.

7. (*a*) Vos, T. A.: La cataracte de la dystrophie myotonique, Ann. d'ocul. **175**:641-666 (Sept.) 1938. (*b*) Ravin, A., and Waring, J. J.: Studies in Dystrophia Myotonica, Am. J. M. Sc. **197**:593-609 (May) 1939.

Although its etiology is unknown, Fleischer [8] has shown dystrophia myotonica to be a heredofamilial degenerative disease exhibiting anticipatory signs through several generations before it develops in its entirety in one generation. These anticipatory signs are: frequent instances of celibacy, many childless marriages, high infant mortality rate and cataracts. In the earlier generations cataracts develop during senility, but with each succeeding generation they develop at an earlier age, until in the "myotonic generation" they are definitely presenile. In addition, the family usually undergoes a gradual degeneration in intellectual, social and moral standards.

In the "myotonic generation" the disease commonly becomes apparent between the ages of 20 and 35, but in rare instances it may appear

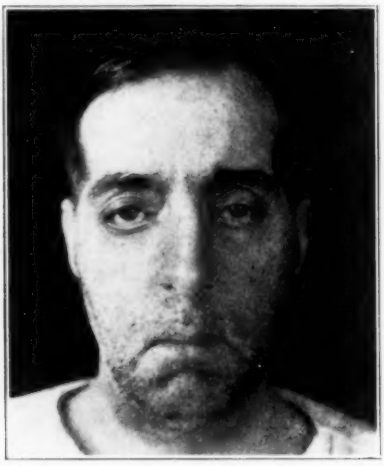

Fig. 1.—Myopathic facies.

earlier. The onset is usually insidious, and the patients are unable to fix a definite time; however, in some cases the symptoms are noticed after a coincidental illness, and occasionally the onset is abrupt.

Myotonia, the slow relaxation of muscular contraction, is almost always present as an early symptom, although a few cases have been reported in which it was absent (Curschmann [9] and Rohrer [10]). It may be elicited by active movement, mechanical stimulation or stimulation with an electric current or by all of these methods. The most common, and at times the only, myotonic manifestation is difficulty in releasing

8. Fleischer, B.: Ueber myotonische Dystrophie mit Katarakt, Arch. f. Ophth. **96**:91-133, 1918.

9. Curschmann, H.: Dystrophia myotonica sine Myotonia, Deutsche Ztschr. f. Nervenh. **74**:157-168, 1922.

10. Rohrer, K.: Ueber Myotonia atrophica (Dystrophia myotonica), Deutsche Ztschr. f. Nervenh. **55**:242-304, 1916.

the hand grasp. However, other isolated muscle groups may be involved, or the distribution may be widespread, affecting all of the muscles until atrophy supervenes. Some of the more usual manifestations of myotonia

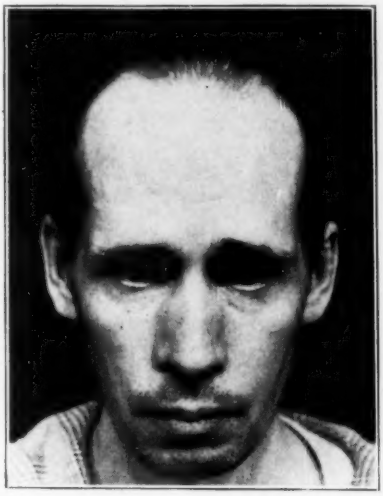

Fig. 2.—Myopathic facies.

Fig. 3 (case 2).—*A*, patient at 16 years of age. The myotonic grasp had been present for four years but myopathic facies had not developed. *B*, patient six years later, with myopathic facies.

are slow speech, stiffness of the jaws, stiffness of the knees and "buckling up" of the feet on beginning to walk or run. Emotion and cold weather seem to intensify the myotonia, and as a result the symptom may vary in any one patient.

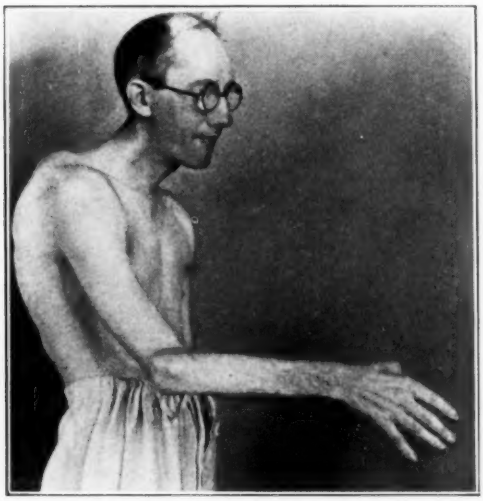

Fig. 4.—Atrophy of the muscles of the forearm and hand.

Fig. 5.—Typical posture and partial baldness and loss of hair.

Atrophy usually is present either at the onset of the disease or before the patient has reached 35 years of age. However, 4 cases have been reported in which atrophy was not present (Rohrer [10] and Fleischer [8]). The muscles of facial expression and mastication, the sternocleidomastoid muscles, the muscles of the forearms, the vastus muscles and the dorsiflexors of the feet are generally the ones in which weakness first appears. Any one or all of these muscles may be affected; however, the orbicularis oculi and sternocleidomastoid muscles are almost always involved. Atrophy of the facial muscles produces a lean, bony, expressionless face with ptosis and tapir mouth (myopathic fascies, figs. 1, 2 and 3 *B*). Involvement of the muscles of mastication results in difficulty in chewing, and weakness of the sternocleidomastoid muscles causes difficulty in raising the head from the prone position. Atrophy of the vastus muscles and the dorsiflexors of the feet result in a broad-based steppage gait.

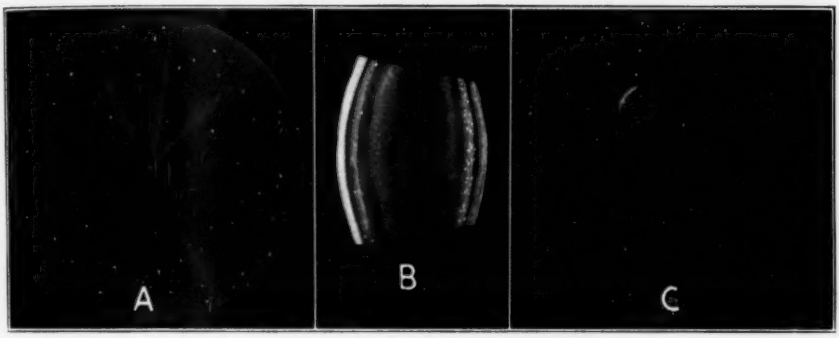

Fig. 6.—Early lens changes: *A*, anterior subcapsular opacities; *B*, schematic optical section of lens; *C*, posterior subcapsular opacities.

If other muscle groups are affected by atrophy, they usually are the small muscles of the hand (fig. 4), the deep muscles of the neck and the muscles of the thighs and upper part of the arms. Progress of the atrophy is variable. It may be slow, and the paralyses may be mild after several years, or it may be rapid, resulting in great disability in one year.

With the exception of cataract, the extramuscular manifestations are less constant than the muscular findings. There may be dryness of the skin, loss of hair, disturbances of the psychic state and libido, mental retardation, atrophy of the testes in men, irregularity of the menses in women and loss of weight. Loss of hair is common and is manifest by baldness and scantiness of general body hair, but it may be limited simply to a high forehead (figs. 2, 4 and 5). The disturbances of the psychic state, as a rule, take the form of reticence, distrust and a defensive attitude, with the patients often resenting examination. The intelligence usually is low, but the patient is shrewd enough to look after his own

interests. The libido may be either depressed or enhanced. The function of the sex glands may remain normal until about 25 or 30 years of age, after which there is atrophy associated with loss of desire and power. Disturbances of other glands of internal secretion are said to occur occasionally.[11] Speech generally is slow, with a nasal quality and lacking intonation. The disease is slowly progressive, but death usually results from intercurrent infection.

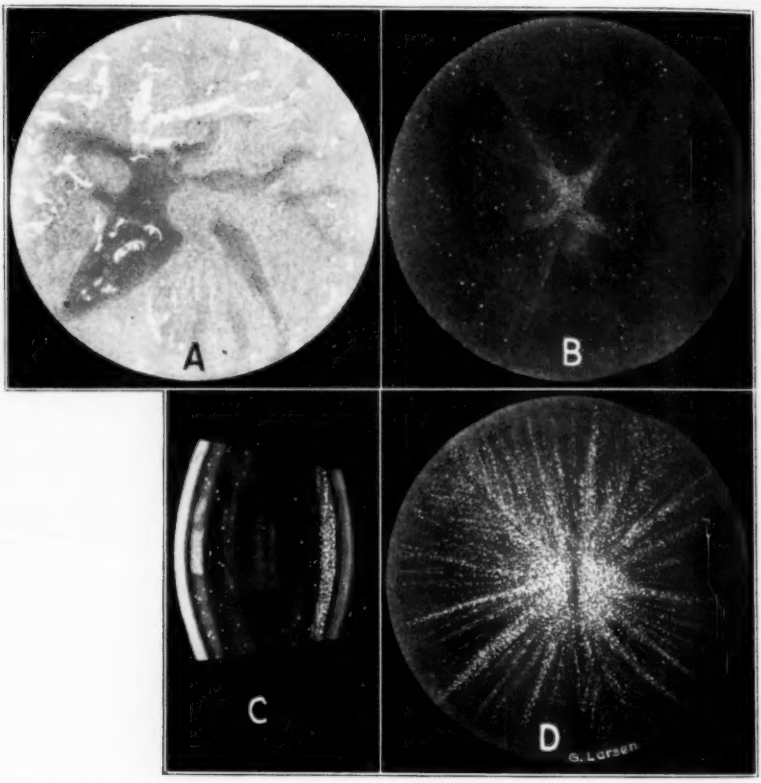

Fig. 7 (case 16).—Lens changes: *A*, mature cataract in the right eye; *B*, anterior subcapsular opacities in the left eye; *C*, schematic optical section of the left eye; *D*, posterior subcapsular opacities in the left eye.

Hoffmann [12] estimated that 10 per cent of patients with dystrophia myotonica had cataract, and Curschmann's [9] estimate was 30 per cent;

11. Adie, W. J., and Greenfield, J. G.: Dystrophia Myotonica (Myotonia Atrophica), Brain **46**:73-127, 1923.

12. Hoffmann, J.: Katarakt bei und neben "atrophischer Myotonie," Arch. f. Ophth. **81**:513-517, 1912.

however, Vogt [13] expressed the belief that all patients would exhibit lenticular changes if examined with the slit lamp and corneal biomicroscope.

The early changes occur in the anterior and posterior cortical regions close to, but not immediately under, the capsule. In these locations numerous, minute, grayish white opacities are interspersed between iridescent crystals of all hues. These opacities and crystals may be diffusely scattered in the superficial cortex (fig. 6), or they may be concentrated along the suture lines and at the anterior and posterior poles of the lens, producing star figures (fig. 7). Occasionally small, clear, bubble-like formations occur in these same cortical layers. The posterior opacities usually occur before, and generally are more advanced than, the anterior ones; however, in one of the cases herein reported the reverse was true.

These changes progress slowly in some cases and more rapidly in others. Fine, dustlike, grayish white opacities and occasional iridescent crystals appear in the deeper layers of the cortex; they are sooner or later followed by water slits, lamellar separations and vacuoles, leading to the development of a complete soft cataract with a small nucleus.

The lenticular changes are bilateral and show relatively the same rate of progress, but as in ordinary senile cataract the changes frequently are more advanced in one eye than in the other (fig. 7).

In the course of eight years, 22 patients with dystrophia myotonica were observed in the State University of Iowa Hospitals. During this same period approximately 110,000 patients were examined, thus making the incidence roughly 1 in 5,000.

All but 3 of the patients were observed on repeated occasions; however, the age and history as recorded in the tables refer to data obtained on first admission.

REPORTS OF CASES

CASE 1.—R. T., a 19 year old white youth, complained of difficulty in releasing his grasp and weakness. The father and sister (T. M., case 3) had identical trouble. General examination revealed atrophy and weakness of the facial muscles, with ptosis and tapir mouth; atrophy and weakness of the forearms and hands with myotonic grasp; sluggish reflexes, and slow nasal speech. Although he complained of no visual disturbance, early lenticular changes were observed on biomicroscopic examination. These consisted principally of small posterior subcapsular opacities and iridescent crystals, with a few anterior subcapsular opacities and accentuation of the anterior and posterior Y of each lens (fig. 6).

CASE 2.—H. R., a 22 year old woman, entered the hospital because of injuries received in an automobile accident. However, there was a history of weakness of the grasp and wasting of the muscles of the forearms and legs of ten years' duration and a syphilitic infection of recent origin. A brother (R. R. Jr., case 7),

13. Vogt, A.: Weitere Ergebnisse der Spaltlampenmikroskopie der vorderen Bulbusabschnittes: IV. Präsenile und senile Linsentrubungen, Arch. f. Ophth. **107**: 192-218, 1922.

father (R. R. Sr., case 20), 2 paternal aunts and a paternal great uncle had similar muscular trouble. General examination revealed ptosis and tapir mouth (fig. 3); myotonic grasp, with atrophy and weakness of the muscles of the face, neck, forearms, hands and lower extremities; loss of hair, lordotic posture; shuffling steppage gait, and slow nasal speech. The Wassermann reaction of the blood was positive. No cells were found in the spinal fluid, and the Wassermann reaction was negative. Vogt's coronary cataracts were found on biomicroscopic examination, but in addition there were posterior subcapsular opacities composed of numerous punctate white dots and iridescent crystals.

CASE 3.—T. M., a 23 year old woman (sister of R. T., case 1), complained of weakness of eighteen months' duration, which had increased and had incapacitated her for the previous six months. However, she had had difficulty in releasing her grasp all of her life. General examination revealed typical myopathic facies, mytonic grasp, steppage gait and nasal speech associated with atrophy and weakness of the facial muscles, sternocleidomastoid muscles and the muscles of the forearms and hands. Vision was found to be 6/21 in the right eye and 6/12 in the left eye. Many punctate white opacities, numerous iridescent crystals and several small vacuoles were observed in the anterior subcapsular regions on biomicroscopic examination. There were a few posterior subcapsular opacities.

CASE 4.—M. McG., a 24 year old woman, reported to the ophthalmic clinic because of blepharitis, but she had been weak and had had difficulty in releasing her grasp for three years. One brother (T. McG., case 5) had similar trouble; another brother "was not strong" but had no definite symptoms. On general examination myopathic facies, myotonic grasp and typically distributed muscular atrophies were found. Although there was no history of visual disturbance and vision was 6/6 in each eye, there were small anterior subcapsular opacities and more advanced posterior subcapsular opacities composed of punctate white dots and small iridescent crystals in each lens.

CASE 5.—T. McG., a 25 year old man (brother of M. McG., case 4), entered the clinic because of blepharitis, but he had typical myopathic facies. After being questioned, he admitted reluctantly that he had been weak and had had difficulty in releasing his grasp for approximately four years. In addition to the facies, examination disclosed myotonic grasp; atrophy and weakness of the sternocleidomastoid muscles and the muscles of the hands, forearms and lower extremities; loss of reflexes; steppage gait, and lordosis. Although vision was normal, biomicroscopic examination revealed scattered punctate white opacities and iridescent crystals in the anterior subcapsular regions with similar but more pronounced changes in the posterior subcapsular zones.

CASE 6.—N. H., a 25 year old man, complained of weakness of three years' duration. One brother (case 10) had similar trouble, and an older brother had cataracts. The patient was found to have a receding hair line, with thinness of the hair; myopathic facies; atrophy and weakness of the sternocleidomastoid muscles and the muscles of the forearms, hands and lower extremities; myotonic grasp; steppage gait, lordosis, and slow nasal speech. On biomicroscopic examination, numerous punctate white opacities and iridescent crystals were found in the anterior and posterior subcapsular zones.

CASE 7.—R. R. Jr., a 27 year old man (brother of H. R., case 2, and son of R. R. Sr., case 20), complained of difficulty in releasing his grasp and general weakness all of his life. Examination revealed a thin, distrustful man with moderate loss of hair, ptosis, tapir mouth, slurred nasal speech, myotonic grasp, steppage gait, lordosis and the typical distribution of muscular atrophy and weakness. Biomicroscopic examination disclosed numerous punctate white opacities and iridescent

crystals in the posterior subcapsular region, with a few similar opacities in the anterior subcapsular zone and an occasional water slit in the cortex of each lens.

CASE 8.—B. M., a 30 year old woman, complained of loss of vision of two years' duration. However, this history was considered unreliable because the patient seemed to have an absolute disregard for truth; e. g., it was only after considerable interrogation that she admitted having an obvious abdominal enlargement. Physical examination revealed a weak, undernourished white woman with a pale, dry skin, typical myotonia and atrophy, steppage gait and lordosis. Vision was limited to perception of light in each eye, and biomicroscopic examination revealed mature cataracts with many iridescent crystals beneath the anterior capsules. The patient refused treatment for the abdominal mass and insisted on cataract extraction. An extracapsular extraction was done, but the patient died before she could return for refraction. Although a postmortem examination was not allowed, the attending physician reported that the patient died of uremia, probably the result of the abdominal tumor.

CASE 9.—J. B., a 31 year old man, entered the hospital because of loss of vision of four years' duration. He had had a cataract extracted from the left eye two years before admission and had had good vision for approximately six months after the operation. For five years he had noticed gradually progressing weakness of his legs, forearms and hands associated with difficulty in releasing his grasp. His mother had had cataracts removed at approximately 24 years of age, and she had had similar weakness of the legs and arms. She had died at 54 of "kidney trouble." General examination disclosed partial baldness; slight ptosis; tapir mouth; expressionless face; inability to wrinkle the forehead or whistle; atrophy and weakness of the sternocleidomastoid muscles and the muscles of the forearms, hands (fig. 4) and legs; myotonic grasp; slow tonic contracture of the muscles when struck with a reflex hammer; loss of deep reflexes; lordosis (fig. 5), and steppage gait. Biomicroscopic examination revealed an incipient cataract of the right eye and a dense after-cataract in the left eye. The changes in the right lens consisted of punctate white opacities and iridescent crystals throughout all layers, a few subcapsular vacuoles anteriorly and lamellar separations in the cortex. The after-cataract was needled, and an extracapsular cataract extraction done on the right eye. Vision was improved to 6/15 in the left eye, but the patient should have a needling in the right eye.

CASE 10.—H. H., a 33 year old man (brother of N. H., case 6), complained of difficulty in releasing his grasp and weakness of the muscles of his forearms and legs of ten years' duration. On general examination there was only slight loss of facial expression; moderate loss of hair; slight ptosis and protruding lips of the tapir type; atrophy with weakness of the sternocleidomastoid muscles and the muscles of the forearms, hands and legs; myotonic grasp; relatively normal reflexes, gait and posture, and average intelligence, his intelligence quotient being 109. Although he complained of no visual loss, changes were found in the lenses with the biomicroscope and slit lamp. There were numerous punctate white opacities and iridescent crystals forming star figures in the anterior and posterior subcapsular regions, with a few water slits in the cortex of each lens.

CASE 11.—C. G., a 35 year old man, complained of difficulty in releasing his grasp and weakness of six years' duration. One brother had similar trouble, but 3 brothers were unaffected. General examination revealed a thin, partially bald, extremely suspicious troublesome man, with nasal speech; blank expression; ptosis;

tapir mouth (fig. 2); atrophy and weakness of the sternocleidomastoid muscles and the muscles of the forearms, hands and legs; myotonic grasp; slow tonic contracture of muscles when hit with a reflex hammer; feeble reflexes; lordosis; steppage gait, and atrophic testicles. Vision was 6/6 in each eye, but biomicroscopic examination disclosed punctate grayish white opacities and iridescent crystals in the anterior subcapsular zones. In the course of observation at intervals over a period of six years, the vision diminished to 6/12 in each eye as the lenticular changes increased.

CASE 12.—M. H., a 36 year old housewife, complained of weakness and difficulty in releasing her grasp of twelve years' duration. One brother had similar trouble; 1 brother had died from tuberculosis, and a sister had died from carcinoma of the stomach. Five siblings were healthy, and the patient had 2 apparently normal children. The physical findings were characteristic, including myopathic facies, muscular atrophies, active and mechanical myotonia, steppage gait, loss of reflexes and thick nasal speech. A complete ocular examination was not made, as the patient became apprehensive and left the hospital a few hours after entering the neurologic clinic.

CASE 13.—L. A., a 38 year old housewife, complained of weakness of ten years' duration. Her father (case 22) had similar trouble. There were 2 normal sisters, and the patient had 1 apparently normal child. Physical findings included atrophy of the muscles of facial expression, with ptosis and tapir mouth and inability to raise the head from a prone position; atrophy and weakness of the muscles of the forearms, hands and legs; myotonic grasp; lordotic posture; steppage gait; diminished reflexes, and nasal speech. Although there was no visual loss, there were numerous punctate white opacities and iridescent crystals in the posterior subcapsular region of each lens.

CASE 14.—D. C., a 38 year old man, entered the hospital because of weakness and diminishing libido of four years' duration. He had four healthy siblings, and among 51 other relatives 1 nephew had similar trouble. The patient was married and divorced, remarried to the same woman and divorced because of a question of legitimacy of 5 children. Examination revealed a thin, partially bald, expressionless man with ptosis and tapir mouth; atrophy and weakness of the sternocleidomastoid muscles and the muscles of the forearms, hands and legs, diminished reflexes (achillis tendon reflex, knee jerk and biceps jerk); lordotic posture; myotonic grasp; steppage gait, and atrophic testicles. The pupillary reaction to light was slow, and there were star-shaped posterior subcapsular opacities in each lens. These opacities were composed of minute white dots and flakes and numerous iridescent crystals. This patient was observed intermittently for six years, during which there was slow progress of the myotonic signs and symptoms. The lenticular opacities slowly became more numerous in the posterior subcapsular region, and new opacities appeared in the anterior subcapsular zones. Vision diminished from 6/6 to 6/9 in each eye during this period.

CASE 15.—R. V., a 41 year old man, entered the hospital because of progressive loss of vision and weakness of ten years' duration. He was married and had 3 healthy children, but a nephew had similar muscular disturbances. The patient was thin and moderately bald, with a blank expression, ptosis and tapir mouth. There was atrophy of the sternocleidomastoid muscles and the muscles of the forearms, hands and legs, associated with myotonic grasp, lordotic posture, steppage gait, loss of deep reflexes, atrophy of the testicles and nasal speech. Vision was limited to perception of light in the right eye and was 6/60 in the left eye. Both

lenses were cataractous, but the condition in the right was more advanced than that in the left. The cataract in the right eye was mature, with numerous iridescent crystals beneath the capsule; that in the left eye was practically complete, with grayish white opacities and numerous iridescent crystals in all layers. An extraction was done on the right eye, and four months later, after some increase in the opacities, an extraction was done on the left eye. Vision for distance with correction before each eye was 6/6, and with correction for near vision the patient could read Oliver 0.50 type at 30 cm.

CASE 16.—C. J., a 45 year old man, was admitted to the hospital because of progressive loss of vision of eight years' duration. He had been weak and had had difficulty in releasing his grasp for one year. He was an obstinate, suspicious, thin, bald man, with a dry skin, thick nasal speech and typical muscular manifestations. The vision was reduced to perception of light in the right eye and was 6/30 in the left. There was a mature cataract of the right lens with many iridescent crystals just beneath the anterior capsule. The cataract in the left eye was not as advanced as that in the right. There was a small star-shaped opacity in the anterior subcapsular region and a larger opacity of the same type posteriorly. These were composed of many fine opacities and numerous iridescent crystals. Lamellar separations, punctate opacities and iridescent crystals occurred throughout the cortex (fig. 7). A cataract extraction was done on the right eye and three months later on the left eye. With correction, after operation the patient could read Oliver 0.50 type at 30 cm., and vision for distance was 6/6 in each eye.

CASE 17.—J. S., a 48 year old woman, was admitted to the hospital because of loss of vision of five years' duration. A cataract operation had been done on the right eye three years previously, but the vision had failed gradually again during the succeeding year. There was a history of weakness and of difficulty in releasing the gasp of five years' duration. The patient was a reticent, thin woman with low intelligence and nasal speech. The skin was dry, and the expressionless face was characterized by ptosis and tapir mouth. There were atrophy and weakness of the sternocleidomastoid muscles as well as the muscles of the forearms, hands and legs, associated with a myotonic grasp, steppage gait, lordotic posture and loss of deep reflexes. The vision was reduced to perception of light in each eye. There was a dense after-cataract in the right eye. In the left lens there were many grayish white opacities of various sizes and numerous iridescent crystals throughout all layers. The after-cataract was needled, and six days later a cataract extraction was done on the left eye. The ptosis was so great that lid crutches had to be built on to the frames, but with this support and with correcting lenses the patient read Oliver 0.75 type at 30 cm. with each eye, and the distance vision was 6/9 in the right eye and 6/6 —3 in the left eye.

CASE 18.—A. G., a 49 year old man, complained of weakness of the upper and lower extremities and difficulty in releasing the grasp of fifteen years' duration. Examination revealed a thin, distrustful man with dry skin, tapir mouth, ptosis and ectropion (fig. 1). There was atrophy of the sternocleidomastoid muscles and the muscles of the forearms, hands and legs. There was slow myotonic relaxation of all muscles after stimulation with a reflex hammer. The deep reflexes were feeble, and the superficial ones were absent. There was a typical myotonic grasp, steppage gait, lordotic posture and indistinct nasal speech. The vision in each eye was 6/6. There were numerous small grayish white opacities and iridescent crystals in the most superficial layers of the anterior cortex of each lens. These opacities were arranged in a star-shaped formation. Similar but more advanced

changes were present in the posterior subcapsular region of each lens. In the course of three years' observation the vision diminished to 6/15 in the right eye and 6/12 in the left eye as the opacities and iridescent crystals increased.

TABLE 1.—*General Manifestations of Dystrophia*

Case, Sex	Age, Marital State	Children	Family	Visual Loss	Myotonic Symptoms	Muscular Findings							
									Myotonia		Atrophy‡		
						Facies	Gait	Posture	Grasp	Other Involvements	Facial Muscles	Sternocleidomastoid Muscles	Muscles of Forearm and Hand
1 ♂	19 Single	0	M C†	0	Entire life	Myopathic	Typical	Normal	Typical	Tongue	++	+	+
2 ♀	22 Single	0	M C	0	10 yr.	Myopathic	Typical	Lordosis	Typical	Tongue	++	++	++
3 ♀	23 Married	0	M C	0	Entire life	Myopathic	Typical	Normal	Typical	Widespread	++	++	++
4 ♀	24 Single	1	M	0	3 yr.	Myopathic	Normal	Normal	Typical	None	+	+	None
5 ♂	25 Single	0	M	0	4 yr.	Myopathic	Typical	Lordosis	Typical	Widespread	++	++	++
6 ♂	25 Single	0	M C	0	3 yr.	Myopathic	Typical	Lordosis	Typical	Tongue	+++	+	++
7 ♂	27 Single	0	M C	0	Entire life	Myopathic	Typical	Lordosis	Typical	Tongue	+++	+	++
8 ♀	30 Single	0	0	2 yr.	? ?	Myopathic	Typical	Lordosis	Typical	Widespread	++++	+++	++++
9 ♂	31 Married	0	M C	4 yr.	5 yr.	Myopathic	Typical	Lordosis	Typical	Widespread	++++	+++	++++
10 ♂	33 Single	0	M C	0	10 yr.	Myopathic	Normal	Normal	Typical	None	+++	++	++
11 ♂	35 Single	0	M	0	6 yr.	Myopathic	Typical	Lordosis	Typical	Widespread	++	++	++
12 ♀	38 Married	2	M	0	12 yr.	Myopathic	Typical	Normal	Typical	Widespread	+	++	+
13 ♀	38 Married	1	M C	0	10 yr.	Myopathic	Typical	Lordosis	Typical	None	++	++	++
14 ♂	38 Divorced	5*	M	0	4 yr.	Myopathic	Typical	Lordosis	Typical	Tongue	++	++	++
15 ♂	41 Married	3	M	10 yr.	10 yr.	Myopathic	Typical	Lordosis	Typical	Tongue	++++	+++	++++
16 ♂	45 Divorced	1	0	8 yr.	1 yr.	Myopathic	Typical	Lordosis	Typical	Widespread	++++	+++	++++
17 ♀	48 Single	0	0	5 yr.	5 yr.	Myopathic	Typical	Lordosis	Typical	Widespread	++++	+++	++++
18 ♂	49 Divorced	3	0	0	15 yr.	Myopathic	Typical	Lordosis	Typical	Widespread	++	++	++
19 ♂	49 Single	0	0	0	10 yr.	Myopathic	Typical	Lordosis	Typical	Tongue	++	++	++
20 ♂	52 Married	3	M C	0	10 yr.	Myopathic	Typical	Lordosis	Typical	Widespread	++	++	++
21 ♀	56 Married	0	0	4 yr.	10 yr.	Myopathic	Typical	Lordosis	Normal	Tongue	++++	+++	++++
22 ♂	60 Married	3	M C	10 yr.	20 yr.	Myopathic	Typical	Lordosis	Typical	Tongue	++	++	++

* Questionable legitimacy.
† M indicates myotonia and C, cataract.
‡ The plus signs indicate the relative degree of atrophy.

CASE 19.—D. R., a 49 year old filling station attendant entered the hospital complaining of weakness of his hands of ten years' duration and weakness of his legs of three years' duration. He had been unable to work for nine months. There

was no history of similar trouble or of presenile cataracts in the family. Examination revealed a well developed, bald, white man with typical myotonic facies and slurred nasal speech. There was atrophy of the sternocleidomastoid muscles and the

Dystrophia Myotonica

			Reflexes		Extramuscular Findings								
	Muscles of Forearm and Hand	Muscles of Legs	Deep	Superficial	Lens Changes	Hair Loss, %	Skin	Speech	Menses	Testes	Mental Attitude	Intellect (I. Q.)
	+	++	Feeble	Normal	Cataract	0	Normal	Typical	.....	Normal	Poor	Normal
	++	++	Absent	Normal	Cataract	25	Dry	Typical	Regular	.....	Poor	Dull (72)
	++	+	Absent	Normal	Cataract	0	Normal	Typical	Regular	.....	Poor	Dull
	None	None	Normal	Normal	Cataract	0	Normal	Normal	Regular	.....	Fair	Dull (84)
	++	++	Absent	Absent	Cataract	10	Normal	Typical	.....	Normal	Poor	Low normal (92)
	++	+	Normal	Normal	Cataract	10	Normal	Typical	.....	Normal	Bad	Dull (77)
	++	++	Feeble	Feeble	Cataract	25	Normal	Typical	.....	Normal	Poor	Dull
	++++	++++	Feeble	Feeble	Cataract	50	Dry	Typical	Irregular	.....	Bad	Stupid
	++++	++++	Absent	Absent	Cataract	50	Normal	Typical	.....	Atrophy ++	Poor	Dull (75)
	++	++	Normal	Normal	Cataract	50	Normal	Normal	.....	Normal	Normal	Norm. (109)
	++	++	Feeble	Feeble	Cataract	75	Normal	Typical	.....	Atrophy ++	Poor	Dull
	+	+	Absent	Absent	Not examined	0	Normal	Typical	Irregular	.....	Bad	Dull
	++	++	Feeble	Absent	Cataract	0	Normal	Normal	Regular	.....	Fair	Dull
	++	++	Feeble	Feeble	Cataract	50	Dry	Typical	.....	Atrophy +++	Fair	Dull
	++++	++++	Absent	Absent	Cataract	50	Dry	Typical	.....	Atrophy ++	Fair	Dull
	++++	++++	Absent	Absent	Cataract	50	Dry	Typical	.....	Atrophy +	Poor	Stupid
	++++	++++	Absent	Absent	Cataract	50	Dry	Typical	Absent	.....	Poor	Dull
	++	++	Feeble	Absent	Cataract	25	Dry	Typical	.....	Normal	Poor	Dull
	++	++++	Absent	Absent	Cataract	75	Dry	Typical	.....	Atrophy +++	Fair	Dull
	++	++	Feeble	Feeble	Cataract	75	Dry	Typical	.....	Normal	Poor	Dull
	++++	++++	Absent	Absent	Cataract	50	Dry	Typical	Irregular	.....	Poor	Dull
	++	++	Feeble	Feeble	Cataract	90	Dry	Typical	.....	Atrophy ++++	Poor	Dull

muscles of the forearms, hands and legs. Superficial and deep reflexes were absent, and the patient was unable to stand without crutches. Even with this support his gait was uncertain and of the steppage type. The grasp was myotonic, and the testicles were atrophic. Vision in the right eye was 6/60 and in the left eye 6/15 —1. With a —1.50 D. sph. ⌒ —0.25 D. cyl., ax. 30 before the right eye and —1.00 D. sph. before the left eye vision was 6/9, and the patient was able

to read Oliver 0.50 type at 32 cm. with a + 1.75 D. sph. addition before each eye. On examination with an ophthalmoscope fine, dustlike opacities were seen in the anterior and posterior layers of the lens. Examination with the slit lamp and biomicroscope revealed numerous punctate white opacities and many iridescent crystals in the superficial layers of the cortex both anteriorly and posteriorly.

CASE 20.—R. R. Sr., a 52 year old man (father of H. R. [case 2] and R .R. Jr. [case 7]), complained of weakness and stiffness of his hands of ten years' duration. In addition to the son and daughter (included in this report), 2 sisters and 1 uncle had similar trouble. The patient was an indolent, troublesome, thin, bald man with dry skin, ptosis and tapir mouth. He had a myotonic grasp, lordotic posture, steppage gait, nasal speech and feeble deep reflexes, associated with atrophy of the sternocleidomastoid muscles and the muscles of the forearms, hands and legs. Vision was 6/6 in each eye, but biomicroscopic examination disclosed punctate white opacities and iridescent crystals in the anterior and posterior subcapsular zones and in the superficial layers of the cortex with an occasional lamellar separation in each lens. The opacities increased in number, but vision remained 6/6 in each eye during the following year.

CASE 21.—M. W., a 56 year old widow, was admitted to the hospital because of progressive loss of vision of four years' duration and weakness of ten years' duration. Examination revealed a thin woman with approximately 50 per cent loss of hair, dry skin, tapir mouth and ptosis. There was advanced atrophy of the sternocleidomastoid muscles and the muscles of the forearms, hands and legs, with loss of the achilles tendon reflex, knee jerk and abdominal reflexes. There was a lordotic posture, steppage gait and nasal speech. Vision was 6/60 in the right eye and was reduced to perception of light in the left eye. There were opacities, iridescent crystals and water slits throughout all layers of the right lens. The left lens was completely opaque and swollen. A cataract extraction was done on the left eye and two months later on the right eye. With correction, the patient was able to read Oliver 0.50 type at 30 cm., and distance vision was 6/6 —1 with the right eye and 6/6 —3 with the left eye.

CASE 22.—H. M., aged 60 (father of L. A., case 13), complained of weakness of twenty years' duration and loss of vision of ten years' duration. The patient was a critical, thin, bald, old man with a dry skin, nasal speech, steppage gait and typical distribution of myotonia and atrophy. Vision was 6/15 in each eye as the result of numerous punctate opacities and iridescent crystals with a few water slits throughout the cortex of each lens.

COMMENT

The 22 cases represented 16 families in which there were histories of 9 additional cases of dystrophia myotonia, numerous instances of senile cataract and 1 case of presenile cataract without muscular symptoms. In family A (cases 1 and 3) a 19 year old boy and his 23 year old sister described similar symptoms and early loss of vision in their father. In family B, 5 members of the family were observed—the father (case 20), the unaffected mother and daughter and an affected daughter (case 2) and son (case 7). In addition to these, there was a history of myotonia and cataracts in 2 sisters and an uncle of the father (fig. 8). In family C, a brother (case 4) and sister (case 5) were observed. An older brother was said to have been weak, but no definite symptoms were elicited:

therefore, he was not considered as having the condition. In family D, 2 brothers (cases 6 and 10) were affected, and an older brother had presenile cataracts without muscular symptoms (fig. 8). In family H, father and daughter (cases 22 and 13) were involved. In family O, mother and son (case 9) had similar symptoms.

The patients' ages ranged from 19 to 60 years, with the average 36. Myotonic symptoms occurred before the thirtieth year in 13 cases

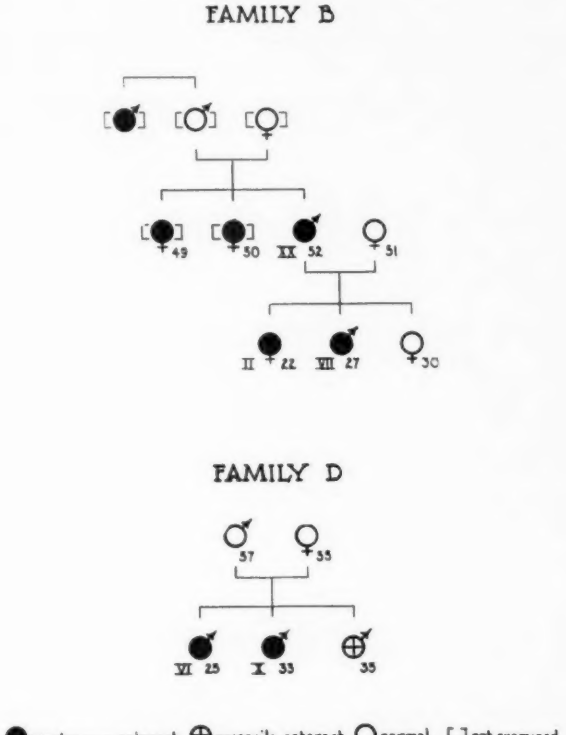

Fig. 8.—Pedigree of two families.

and before the forty-sixth year in the remainder. The duration of symptoms varied from one to twenty years, the average being ten years.

Seven, or one third, of the patients entered the hospital because of loss of vision. Two others were seen in the ophthalmic clinic because of chronic blepharoconjunctivitis, and a diagnosis of dystrophia myotonica was made as a result of slit lamp evidence of lenticular changes together with subsequent neurologic findings. Thirteen, or 59 per cent, were examined first in the department of neurology because of "muscular weakness." Twelve of these patients were referred to the ophthalmic

clinic for examination, but the thirteenth (case 12) became unduly alarmed and left the hospital before her eyes could be examined completely. In our experience reactions of this type were not uncommon.

Lenticular changes were observed in all cases (21) in which slit lamp examination was made (table 2), and these changes were seen to increase with time. Some of the earlier lenticular changes were not visible with an ophthalmoscope, but as the disease progressed fine, dustlike opacities were observed with this instrument. The earliest changes occurred

TABLE 2.—*Visual Loss and Lenticular Changes at First Examination*

			Visual Loss		Lenticular Changes *	
Case	Age	Sex	O. D.	O. S.	O. D.	O. S.
1	19	♂	0	0	a-c-i-P	a-c-i-P
2	22	♀	0	0	P-i	P-i
3	23	♀	6/21	6/12	A-i-V	A-i-V
4	24	♀	0	0	a-i-P	a-i-P
5	25	♂	0	0	a-i-P	a-i-P
6	25	♂	0	0	a-i-P	a-i-P
7	27	♂	0	0	a-i-c-l-P	a-i-c-l-P
8	30	♀	LP †	LP	Mature cataract-i	Mature cataract-i
9	31	♂	LP	LP	A-v-c-l-i-P	After-cataract
10	33	♂	0	0	a-i-l-P	a-i-l-P
11	35	♂	0	0	a-i-P	a-i-P
12	38	♀	0	0	?	?
13	38	♀	0	0	P-i	P-i
14	38	♂	0	0	P-i	P-i
15	41	♂	LP	6/60	Mature cataract-i	A-C-i-l-P
16	45	♂	LP	6/30	Mature cataract-i	A-c-i-l-P
17	48	♀	LP	LP	After-cataract	A-C-i-l-P
18	49	♂	0	0	a-i-P	a-i-P
19	49	♂	6/9	6/9	a-i-c-P	a-i-c-P
20	52	♂	0	0	a-c-i-l-P	a-c-i-l-P
21	56	♀	6/60	LP	A-c-i-l-P	Intumescent cataract
22	60	♂	6/15	6/15	A-c-i-l-P	A-c-i-l-P

* The principal changes are in capitals. The letters indicate: a, anterior subcapsular opacities; c, cortical opacities; i, iridescent crystals; l, lamellar separation (water slits); P, posterior subcapsular opacities, and V, vacuoles. The question mark means that the patient was not examined with a biomicroscope.

† LP means perception of light.

posteriorly and consisted of minute white opacities and iridescent crystals situated in the most superficial layer of the cortex and concentrated in a stellate manner. There was one exception (case 3), in which the early changes occurred anteriorly and vacuoles appeared at the anterior pole in the same layer with the white opacities and iridescent crystals. However, in the other cases the posterior opacities and crystals increased slowly in number and were followed later by similar changes in the anterior superficial cortical layer. Later deep cortical opacities and lamellar separations appeared, but none was observed to develop rapidly into a soft cataract. One patient (case 18) was observed at intervals

for three years. In that time his lenticular changes progressed and his vision gradually diminished from 6/6 in each eye to 6/15 in the right eye and 6/12 in the left eye. In another instance (case 11) the vision gradually diminished from 6/6 to 6/12 in each eye in the course of six years. In the 7 cases of soft and mature cataracts loss of vision had been noticed for two to ten years, but in all cases at least two years elapsed between the first symptom of loss of vision and loss of all useful vision. Furthermore, these 7 patients exhibited more advanced neuromuscular symptoms than the other patients. Therefore, there apparently was some correlation between the development of cataracts and other symptoms, as slowly developing muscular signs were accompanied by a slowly developing cataract and rapidly developing muscular symptoms were accompanied by earlier development of a mature cataract.

TABLE 3.—*Results of Cataract Extraction*

Case	Eye	Vision	Condition of Lens	Operation	Result (Corrected)
8	O. D.	LP*	Mature	Extraction	Deceased before correction
	O. S.	LP	Mature		
9	O. D.	LP	Premature	Extraction	To return for needling
	O. S.	LP	After cataract	Needling	6/15
15	O. D.	LP	Mature	Extraction	6/6 Oliver 0.50 at 30 cm.
	O. S.	6/60	Premature	Extraction	6/6-1 Oliver 0.50 at 30 cm.
16	O. D.	LP	Mature	Extraction	6/6 Oliver 0.50 at 30 cm.
	O. S.	6/30	Premature	Extraction	6/6 Oliver 0.50 at 30 cm.
17	O. D.	LP	After cataract	Needling	6/9 Oliver 0.75 at 30 cm.
	O. S.	LP	Premature	Extraction	6/6-3 Oliver 0.75 at 30 cm.
21	O. D.	6/60	Premature	Extraction	6/6-1 Oliver 0.50 at 30 cm.
	O. S.	LP	Intumescent	Extraction	6/6-3 Oliver 0.50 at 30 cm.

* LP indicates perception of light.

Similar lenticular changes have not been observed in other types of the chronic progressive muscular dystrophies examined during this period of observation.

Other ocular manifestations of the disease are ptosis, loss of orbital fat and ectropion. Ptosis was variable in degree but present in each of the 22 cases. In 2 of the cases it was so great that special spectacles with "lid crutches" were required after cataract extraction. In several cases loss of orbital fat was observed as a part of general emaciation, and this factor combined with ptosis tended to simulate enophthalmos. Ectropion was observed in 1 case, but it was of mild degree even though the patient had had muscular symptoms for fifteen years. Paralysis of ocular rotations was not observed in this group and has not been reported. Diplopia has not been described as a symptom of this disorder. Although Lohlein [14] reported 1 case of yellowish atrophy of one

14. Lohlein, W.: Frühkatarakt bei atrophischer Myotonie, Klin. Monatsbl. f. Augenh. **52**:453-457, 1914.

nerve head associated with myotonia atrophica, fundus lesions have not been reported, and no significant lesions of the fundus or nerve head were observed in these patients.

Cataract extraction, therefore, is not contraindicated by other ocular pathologic involvement incidental to this disease and usually results in good visual acuity (table 3).

CONCLUSIONS

1. Lenticular changes were observed in each case of myotonia atrophica in which biomicroscopic examination was done even though several of the patients had experienced no visual disturbances.

2. These opacities were observed to increase on repeated examinations.

3. Among the other ocular manifestations of the disease, ptosis is almost constantly present; ectropion is present occasionally, and in some instances enophthalmos is simulated because of the loss of orbital fat.

4. In spite of the fact that 22 cases were observed in eight years, dystrophia myotonica is a rare disease, but probably not as rare in the United States as one might suppose from the small number of case reports in the literature.

HEREDITARY PRIMARY GLAUCOMA

A PEDIGREE WITH FIVE GENERATIONS

WILLIAM H. STOKES, M.D.
OMAHA

Although primary glaucoma is relatively common in its occurrence, it is difficult to state with any degree of certainty how often the disease may be hereditary. In 1906 Nettleship [1] stressed the need for the collection of more data relating to the hereditary nature of glaucoma. Statistics have accumulated slowly, and the evidence is still too vague to justify definite conclusions. A large proportion of the published pedigrees include notes on not more than two generations, and in these only 2 or 3 patients have been studied. Although such records are useful for statistical purposes in respect to sex and age and for the purpose of illustrating certain features of individual case records, they are, unfortunately, of little value in regard to the special study of hereditary factors.

In 1869 von Graefe [2] emphasized the importance of inheritance in the etiology of glaucoma and referred to families in which the disease had occurred in three or four generations, but he did not actually report pedigrees. The most exhaustive study of this subject was made by Julia Bell [3] in 1932 in the Nettleship memorial volume. The inheritance of glaucoma was traced into two generations in 48 of the 68 pedigrees collected by Bell; three or more successive generations were affected in 20 pedigrees reviewed. As far as I am able to determine at present, there exist 27 pedigrees in which the disease has been traced in members of three or more generations, for many of which, however, no clinical description is given (table). For this reason, therefore, only tentative suggestions can be made from a review of the literature.

AGE OF ONSET AND SEX INCIDENCE

In 1932 Bell [3] wrote that the age of onset in recorded cases of hereditary primary glaucoma showed a marked contrast to that in records

1. Nettleship, E.: Some Hereditary Diseases of the Eye, Ophthalmoscope **4**:493-549, 1906.

2. von Graefe, A.: Beiträge zur Pathologie und Therapie des Glaukoms, Arch. f. Ophth. **15** (pt. 3):228, 1869.

3. Bell, J.: On Some Hereditary Structural Anomalies of the Eye and on the Inheritance of Glaucoma, in Bell, J.: The Treasury of Human Inheritance, London, Cambridge University Press, 1932, vol. 27, pt. 5, p. 448.

Summary of Previously Published Pedigrees of Hereditary Glaucoma Occurring in Three or More Generations

Author	Generation	Members Affected	Age of Onset	Type of Glaucoma	Anticipation	Clinical Findings	Comment
Harlan,[20] 1885	V	Male, 3 Female, 6 Total, 9	Male, 26 Female, 20 Average age, 21	Acute inflammatory	0	Not given	The condition in all cases apparently had an inflammatory onset; the disease made its appearance before the age of 20 in most of the affected members
Howe, L.: Arch. Ophth. **16**: 72, 1887	III	Male, 5 Female, 3 Total, 8	Male, 28 Female, 23 Average age, 26	Acute, 3; subacute or chronic, 5	+	Not given in detail	Out of the 8 persons affected, 4 have been operated on by iridectomy; 5 are totally blind in both eyes and 2 in one eye
Rudin, V.: Klin. Monatsbl. f. Augenh. **60**: 592, 1918	III	Male, 3 Female, 1 Total, 4	Male, 26 Female, 30 Average age, 27	Not stated	0	Not given	Glaucoma was observed in 2 brothers at the ages of 27 and 20 years; father and paternal grandmother became blind at the age of 30
Rogman,[13] 1899	III	Male, 3 Female, 2 Total, 5	Male, 38 Female, 72 Average age, 48	Acute, 2; chronic, 3	+	Not given	Acute glaucoma developed from glaucoma simplex; all persons affected had prodromal symptoms for years before operation (iridectomy)
Nettleship,[1] 1906 (Lawford,[11] 1908)	III	Male, 2 Female, 2 Total, 4	Male, 71 Female, 32 Average age, 45	Chronic, 3	+	Myopia	Further information of the pedigree are given by Lawford in 1908; there were long periods of prodromal symptoms
Fleischer, B.: Verhandl. d. Gesellsch. deutsch. Naturf. u. Aerzte **97**: 250, 1907 (Plocher,[5] 1918)	IV	Male, 2 Female, 4 Total, 6	Male, 32 Female, 22 Average age, 27	Chronic	+	Not given	The pedigree was originally published in 1907; later information was communicated by the recorder to Plocher who published it in 1918
Kauffman, F.: Wchnschr. f. Therap. u. Hyg. d. Auges **11**: 68, 1907	IV	Male, 2 Female, 6 Total, 8	Male, 24 Female, 24 Average age, 24	Not mentioned	0	Not given	This pedigree is of interest because of hereditary data, but the clinical data are lacking
Wallenberg,[27] 1907 (1910)..	IV	Male, 3 Female, 3 Total, 6	Male, 22 Female, 22 Average age, 22	Rapidly progressive; acute onset	0	Not given	This pedigree was republished in 1910; all affected members became blind; the onset occurred at the same period of life in all
Lawford [11] (1908); Roy. London Ophth. Hosp. Rep. **19**: 42, 1914	III	Male, 3 Female, 3 Total, 6	Male, 40 Female, 57 Average age, 51	Chronic	+	Not given; corneal diameters, 12 to 12.5 mm.	This pedigree was first published in 1908; it was republished by Lawford in 1914; the clinical findings are incomplete
Calhoun,[10] 1914	III	Male, 7 Female, 2 Total, 9	Male, 16 Female, 11 Average age, 16	Chronic, simplex type in all	+	Given	The corneal measurements were within normal limits; the anterior chambers were of normal depth; emmetropia was disclosed on refraction; the operation of choice was the Elliot trephination
Plocher,[5] 1918 (Korte,[17] 1939)	VI	Male, 7 Female, 11 Total, 18	Male, 37 Female, 26 Average age, 29	In the majority chronic and occasional inflammatory symptoms	+	Given	A complete family history is given; in 1939 Korte republished the pedigree with additions; refraction of all affected members showed myopia; the anterior chamber varies in depth; consanguinity is revealed by history
Vogt, A.: Arch. f. Ophth. **111**: 91, 1923	III	Male, 3 Female, 4 Total, 7	Onset not given	Bilateral in each case; type (?)	Not given	Not given	Details of cases are not given nor is the age of onset of the disease

Holland, H. T.: Indian M. Gaz. **59**: 408, 1924	III	Male, 3 Female, 1 Total, 4	Onset not given	In none had the onset been acute; gradual loss in vision	Not given	Not given	All members of the family were blind between the ages of 50 and 60; the age of onset is not given
Courtney and Hill [4]	V	Male, 10 Female, 8 Total, 18	Male, 19 Female, 23 Average age, 20	Chronic simplex	Not marked; succeeding generation seen earlier	Given	Corneal measurements show normal diameters; refraction showed practically all members to be emmetropic; the corneas were of normal diameter; the disease could not be influenced by miotics
Shumway [7]	IV	Male, 1 Female, 5 Total, 6	Male, 13 Female, 16 Average age, 16	Chronic, rapid course	0	Not given in detail	All affected members lost their sight with or without operation
Berg [18]	VII	Male, 13 Female, 9 Total, 22	Male, 30 Female, 26 Average age, 27	Chronic	0	Given	Atrophy of the iris was noted in the effected members; depth of the anterior chamber is given; hydrophthalmos developed in 2 of the members
Zorab [22]	III	Male, 6 Female, 1 Total, 7	Male, 42 Female, 8 Average age, 39	Chronic	+	Given in part	Three of the 7 affected members were myopic; refraction is not indicated at all; corneal measurements are not given; the persons operated on retained vision
Neame, in discussion on Zorab [22]	III	Male, 4 Female, 3 Total, 7	Male, 38 Female, 28 Average age, 34	Chronic	0	Given in part	Myopia (small degree) was present; the operation of choice was the Elliot trephine method
Waardenburg [34]	III	Male, 4 Female, 1 Total, 5	Not given	Subacute and chronic	0	0	Clinical details and age of onset are not given; the literature is reviewed and a bibliography given
Kirkpatrick [36]	III	Male, 2 Female, 2 Total, 4	Male, 46 Female, 47 Average age, 46	Chronic	0	Not given	This is apparently the only family of Jewish ancestry
Klein, cited by Bell [3]	III	Male, 0 Female, 3 Total, 3	Male, 0 Female, 35 Average age, 35	Not stated	+	Not given	All (3) affected members were females
Wolfsohn-Jaffe, E.: Klin. Monatsbl. f. Augenh. **94**: 692, 1935	III	Male, 6 Female, 3 Total, 9	Male, 39 Female, 40 Average age, 39	Chronic; inflammatory attacks	0	Given	When the glaucoma was of inflammatory type, iridectomy was performed; only 2 had trephine operations
Biro [28]	III	Male, 4 Female, 4 Total, 8	Male, 25 Female, 32 Average age, 29	Chronic in all	+	Not given	Biro gives 4 family pedigrees; his conclusion: the younger the patient, the more hopeless the treatment
Biro [28]	III	Male, 1 Female, 6 Total, 7	Male, 58 Female, 40 Average age, 49	Inflammatory	+	Not given	Persons in all three generations had acute inflammatory attacks
Biro [28]	III	Male, 1 Female, 4 Total, 5	Not given	Chronic simplex	+	Not given	The author states that glaucoma showed anticipation, but the age is not given in pedigree
Biro [28]	III	Male, 2 Female, 3 Total, 5	Male, 20 Female, 42 Average age, 30	Inflammatory	+	Not given	Both grandparents were glaucomatous
Briggs [10]	III	Male, 6 Female, 4 Total, 10	Not given	Primary	Not given	Not given in detail	The Elliot trephine operation was the method of choice

which include all cases of glaucoma. The average age of onset in the cases of hereditary glaucoma was twenty years earlier than the age in the general series, 52 per cent of cases of hereditary glaucoma occurring before the age of 30 years. The glaucoma in only 3.6 per cent of the 3,021 cases in the general series occurred before the age of 30. These differences cannot be explained entirely by the earlier recognition of the disease in the hereditary type of glaucoma but suggest some fundamental difference in the determining factors of the two groups. There are authors (Courtney and Hill,[4] Plocher,[5] Sattler,[6] Shumway[7] and others) who have classified the hereditary glaucoma in their cases as belonging to the juvenile type, the age incidence being extended to the age of 30 or 35 as an upper age limit for juvenile glaucoma. In this connection I wish to quote Anderson,[8] who in 1939 wrote as follows: "No absolute distinction can be made to separate juvenile glaucoma from hydrophthalmia (congenital glaucoma) on the one hand and senile glaucoma on the other. It is probable that at one end of the scale, that is, in early life, an innate defect in structure is the cause of hypertension and that degeneration is merely a secondary phenomenon. At the other end of the scale, raised tension is due almost entirely to various degenerative changes associated with old age, and inherited defects play a minor part. There is an intermediate group in which both factors appear to be pathogenic in varying degrees." In deciding the lower age limit between juvenile glaucoma and hydrophthalmia, Anderson has taken as a criterion the absence or presence of the most characteristic sign of congenital glaucoma, namely, distention of the eyeball. Thus "glaucoma due to a congenital defect may be classified as juvenile if it fails to develop until the eye is no longer distensible." Ophthalmologists should concern themselves with the following issues, which at present are difficult to determine. If it is taken for granted that the intraocular pressure is always raised in congenital glaucoma (hydrophthalmia), what degree of tension can exist in childhood without causing enlargement of the globe? Can distention occur as a result of increased intraocular pressure in later life, and if so what is the relation between distensibility of the globe and maturity? In my studies of the subject I am led to believe that a more precise definition of the word juvenile is necessary in respect to certain organs and structures when considered from a developmental point of view. In hydrophthalmia the condition was recognized before

4. Courtney, R. H., and Hill, E.: Hereditary Juvenile Glaucoma Simplex, Tr. Sect. Ophth., A. M. A., 1931, pp. 47-68.

5. Plocher, R.: Beitrag zum juvenilen familiären Glaukom, Klin. Monatsbl. f. Augenh. **60**:592-619, 1918.

6. Sattler, R.: Juvenile Glaucoma, Tr. Am. Ophth. Soc. **10** (pt. 3):519, 1905.

7. Shumway, E., in discussion on Courtney and Hill.[4]

8. Anderson, J. R.: Hydrophthalmia or Congenital Glaucoma, London, Cambridge University Press, 1939.

the age of 1 in 87 per cent of the cases in Anderson's [8] series; only 5 patients (2 per cent) were more than 6 years old before their eyes became affected. If hydrophthalmia is to be regarded as the glaucoma of infancy, then a study of the hereditary nature of the disease should be of considerable value. In a large series of recorded cases, however, the hereditary factor in this disease can be demonstrated only in a comparatively few instances. No doubt heredity plays a part and possibly a much greater part in congenital glaucoma than one has been led to believe. So often a defective offspring faced with a future of blindness becomes the last of the family because of a physical handicap which precludes marriage.

In a review of the material on juvenile glaucoma from the ophthalmic department of the University of Leipzig, Keerl [9] found that heredity was a factor in 18 per cent of the cases in this group. The patients in whom juvenile glaucoma occurred were distributed according to age as follows: from 6 to 15 years, 14.8 per cent; from 16 to 25 years, 37 per cent, and from 26 to 35 years 48.3 per cent.

In the 27 pedigrees which I have reviewed, which include a total of 210 patients with glaucoma of a hereditary nature, the average age of onset of the disease was 32 years for the males and 30 years for the females. In the majority of the pedigrees the onset fell between the ages of 20 and 30. In the pedigree cited by Shumway [7] in which 6 members were affected in four generations, the average age of onset was 16 years. Calhoun [10] reported 9 glaucomatous patients in a pedigree of three generations with an average of 16 years. Eighteen descendants in five generations of a family reported by Courtney and Hill [4] had glaucoma at an average age of 20 years. It is of interest to contrast with the pedigree reported by Lawford [11] (Nettleship) in 1908. In 4 males in a sibship [12] of 10 glaucoma developed probably late in life. Their ages at the time of examination were 66, 67, 72 and 77. Since the records of many pedigrees are incomplete and fail to state the age of onset of the disease and the period of the prodromal symptoms, it is obvious that there are possible sources of error in calculating the actual age of onset. The average age of onset of glaucoma in the members of the pedigree which I am reporting is 25 years. In general, glaucoma tends to affect women a little earlier than men, and this also seems to be the case in the hereditary group.

9. Keerl, M.: Das Glaukom der Jugendlichen, Inaug. Dissert., Leipzig, Emil Lehmann, 1920.

10. Calhoun, F. P.: Hereditary Glaucoma (Simplex), Tr. Sect. Ophth., A. M. A., 1914, pp. 101-121.

11. Lawford, J. B.: Examples of Hereditary Primary Glaucoma, Roy. London Ophth. Hosp. Rep. **17**:57-68, 1908.

12. Sibships, siblings and childships are terms introduced to indicate all the offsprings of one parentage.

It seems that the age at which the rise of tension appears varies in the different families, and if all the reported cases of hereditary glaucoma are taken together examples can be found in which the onset occurred at all ages (in juvenile, adolescent, adult and senile persons). Since it is not known what proportion of cases of primary glaucoma are free from genetic potentialities, interest should be aroused in the hereditary tendency of the disease in all cases, but especially so when the disease occurs in the young adult.

The tendency for the disease to appear at an earlier age in succeeding generations is termed "anticipation." The same term is used if the disease appears at an earlier age in the members of the same generation. Von Graefe [2] recognized this tendency to anticipation in hereditary glaucoma, but he stated that he had observed an unusually long prodromal period existing in some patients for ten or fifteen years. Anticipation is emphasized in the pedigrees recorded by Rogman,[13] Nettleship,[1] Lawford,[11] Calhoun,[10] Plocher [5] and others. There are pedigrees in which it is not possible to demonstrate whether this phenomenon does or does not hold. An interesting conclusion reached by Bell [3] was that the age of onset of the disease in the parents bears a close relation to the age of onset in the affected offspring, the children tending to become affected about ten years earlier than the parents. In my own pedigree (chart 1) the 5 members of the third generation were blind at an average age of 20, the 7 members of the fourth generation showed an average onset of the disease at an age of 35 and in the 7 members of the fifth generation the onset of glaucoma occurred at an average age of 21. It seems that members of a sibship tend to become susceptible at the same age. However, the onset of the disease as previously mentioned may be so gradual that its date can seldom be fixed with any approach to accuracy. It is, of course, possible that members of succeeding generations were seen earlier and therefore a diagnosis was made more promptly. For that reason the reduced age of onset in the younger generation must not be taken as proof of a general tendency to anticipation on the transmission of the disease.

The liability to transmission by the two sexes is roughly equal (Lawford,[11] Plocher [5] and Calhoun [10]). Löhlein [14] in his study of 92 glaucomatous patients under 30 years of age (not necessarily hereditary) found a slight predominance of males. Of the 210 glaucomatous persons in the 27 pedigrees which I have examined, 106 were males and 104 females. In my own pedigree (chart 1) there are at the present time 21 affected members in five generations—12 males and 9 females. It seems

13. Rogman, A.: A propos de la pathogénie du glaucome chronique simple, Clin. opht. **5**:73-75, 1899.

14. Löhlein, W.: Das Glaukom der Jugendlichen, Arch. f. Ophth. **85**:393-488, 1913.

that the sex incidence in hereditary glaucoma provides no distinctive feature as compared to that with glaucoma in general. In hydrophthalmia, however, the predominance of males is marked (Anderson [8]).

SIZE OF CORNEA, DEPTH OF ANTERIOR CHAMBER AND REFRACTIVE ERROR

The normal corneal diameter of infants is between 8 and 10 mm. The period of greatest growth occurs during the first year, when the measure-

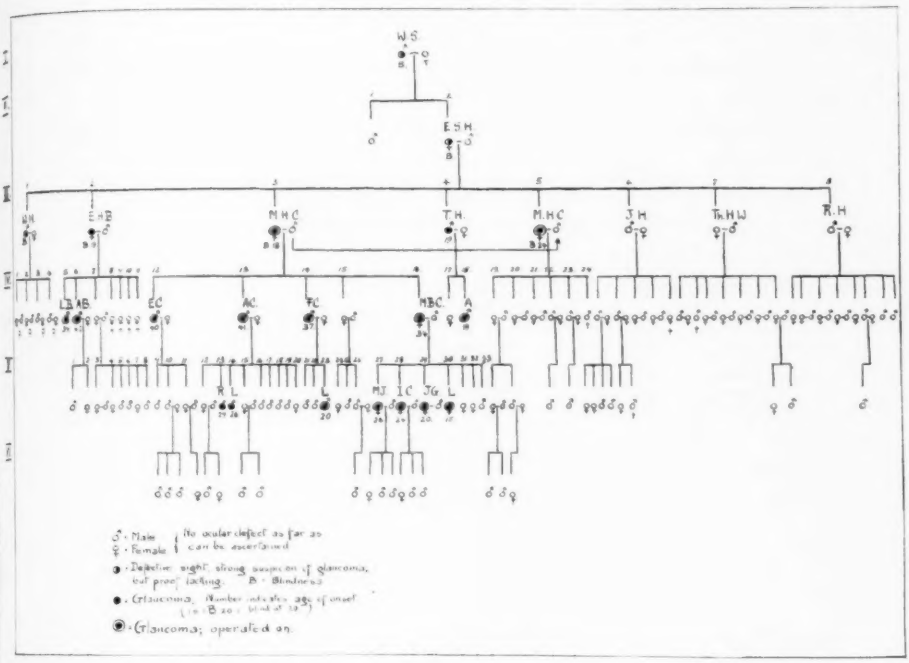

Chart 1.—Pedigree of family with glaucoma in five generations.

ments may be 11.4 mm. Priestly Smith [15] measured the corneas of 1,000 healthy adult eyes and of 216 eyes affected with primary glaucoma. He found that the average horizontal diameter of the corneas of healthy eyes was 11.6 mm. and of glaucomatous eyes 11.2 mm.; while 22.7 per cent of the corneas of glaucomatous eyes measured 10.5 or 10 mm., only 1.7 per cent of healthy eyes had corneas of so small a size. Thus it will be seen that the corneas of the greater number of glaucomatous eyes were within normal limits, but a large number of small corneas were found among glaucomatous patients. Priestly Smith also noted that eyes

15. Smith, P.: On the Pathology and Treatment of Glaucoma, London, J. & A. Churchill, 1891.

which were blind as the result of glaucoma and which had been enucleated were characterized by a small globe, a disproportionately large lens, a shallow anterior chamber and a closed filtration angle. These findings would suggest a source for glaucoma on a hereditary basis. A malproportioned and small globe having a tendency to obstruction of the filtration angle might readily be expected to occur in familial glaucoma. However, such does not seem to be the case. Bell [3] found that there was considerable evidence against this hypothesis. The cornea had been measured or its size estimated in 25 eyes of the series which she reviewed, and of these there were only 2 that could be described as being small.

The corneal measurements have been cited in only a few of the 27 pedigrees which I have examined, and these were of normal proportions, as recorded by Courtney and Hill,[4] Calhoun,[10] Plocher [5] and Briggs.[16] From a review of the literature there is no evidence that hereditary glaucoma is due to some degree of congenital microphthalmia. In my own glaucomatous pedigree the horizontal corneal measurements showed this structure to be between 11.5 to 12.5 mm. in size.

In a questionnaire collected by Anderson [8] in regard to the size of the cornea in hydrophthalmia, reference was made to the corneal measurements of 110 eyes. Of these, only 3 were described as being normal and 56 were described as very large or having a diameter between 14 and 15.75 mm. In 8 instances the corneal diameter measured 16 mm. or more.

In the great majority of reported cases of hereditary glaucoma no statement is made regarding the depth of the anterior chamber. Bell [3] found 32 references to this matter, and of the ones cited there were 13 eyes in which glaucoma developed before the age of 19, in 4 of which the anterior chambers were normal or, as in 1 case, rather deep. In 6 instances the chambers were shallow, and in 1 the chamber was obliterated; 5 of the chambers in the latter group were of the acute, or congestive, type. In the pedigree reported by Calhoun [10] deep anterior chambers were mentioned in his case histories. The author, however, meant to convey the impression that the anterior chambers were not shallow, since as a matter of actual fact they were all of normal depth. Plocher [5] and Korte [17] reported chambers which were usually deeper than normal. In the pedigree reported by Berg [18] the anterior chambers were normal in all or slightly deeper than normal. In my own patients

16. Briggs, A. H.: Familial Primary Glaucoma in Adults, Brit. J. Ophth. **23**:649-659, 1939.

17. Korte, W.: Beiträge zur Erblichkeit des Glaukoms, Klin. Monatsbl. f. Augenh. **102**:664-668, 1939.

18. Berg, F.: Erbliches jugendliches Glaukom, Acta ophth. **10**:568, 1932.

the chambers were of normal depth, averaging about 3.5 mm.[19] The distance from the central portion of the posterior surface of the cornea to the anterior pole of the lens varies somewhat at different ages, measuring according to Meesmann [20] about 3.69 mm. at the age of 15 years and 3.04 mm. at the age of 65 years.

In hydrophthalmia the depth of the anterior chamber is often deep; in fact, it may be very much so—for example, 12 to 18 mm. The average depth was 5.75 mm. in Anderson's [8] series of cases of hydrophthalmia. The deeper anterior chamber may perhaps be explained by the greater plasticity of the tissues in youth which allows the shape to be modified through raised pressure. One wonders how much of the shallowness of the anterior chamber (seen in congestive glaucoma and in glaucoma of old age) is due to secondary inflammatory and degenerative changes and therefore a sequel to, rather than a cause of, increased pressure.

It has been stated that in hydrophthalmia the cornea and the adjacent sclera distend first and then the posterior portion of the sclera. Myopia is the most common refractive condition found in hydrophthalmia, but it is present usually in only a moderate degree (1 to 7 diopters) and is not nearly as marked as the increased length of the eye would suggest. The relation between the length of the axis of the eyeball and the degree of ammetropia, which is nearly constant in myopia, does not hold in hydrophthalmia. Parsons [21] gave three reasons why the hydrophthalmic eye is not nearly so myopic as the axial elongation would indicate: 1. Because of the decreased corneal convexity, the radius of the corneal curvature approximates that of sclera. 2. The same decreased convexity applies to the lens, owing to stretching of the suspensory ligament. 3. The lens is displaced backward.

In myopia the most distended parts are those posterior to the ora serrata, and the most obvious changes are those near the posterior pole. The cornea and anterior chamber usually are not affected. The increase in the anteroposterior diameter of the globe in myopia, however, is not due to distention in the sense that there is an existing increased intraocular pressure.

In the hereditary type of glaucoma, which often affects members of a group when they are between the ages of 15 and 25, a high intraocular pressure no doubt is a factor in the production of myopia because of the elasticity of the eyeball. The onset of the myopia may even prove compensatory to the high tension for a period when there are no pro-

19. Measurements were made with the slit lamp and the corneal microscope.

20. Meesmann, A.: Die Mikroskopie des lebenden Auges an der Gullstrandschen Spaltlampe mit Atlas typischer Befunde, Berlin, Urban & Schwarzenberg, 1927, p. 71.

21. Parsons, J. H.: Refraction in Buphthalmia, Brit. J. Ophth. **4**:211, 1920.

dromal symptoms and no characteristic field changes of glaucoma. It is still uncertain whether the myopia found in hereditary glaucoma is entirely secondary to the increased intraocular tension or whether the relation is one of a correlated inheritance similar to that between myopia and other ocular anomalies. However, the incidence of myopia in hereditary glaucoma is sufficiently great to suggest that the hypertension had been influential in producing the bulbar distension and the altered refraction. In the pedigree published by Plocher [5] and republished by Korte [17] all of the 18 members affected with glaucoma were myopic. In Zorab's [22] pedigree 3 of the 7 affected members were myopic, and the refraction was not indicated for the other 4 members. Nettleship's [23] and Somya's [24] patients were myopic. Löhlein [14] found that approximately 50 per cent of the glaucomatous eyes in the younger group were myopic, and about one third of them had myopia of more than 6 diopters. Only 15 per cent of his patients with senile glaucoma were myopic. Haag's [25] statistics show that 31 per cent of his young glaucomatous patients were myopic and that 17 per cent of them had a myopia of 6 diopters or more. Keerl [9] showed myopia to be present in 50 per cent of his patients, and 21 per cent of them were myopic, 6 diopters or more. In the pedigrees published by Courtney and Hill,[4] Calhoun [10] and others the refraction was practically emmetropic or only slightly myopic. In my own cases, in which the refractive error was determined for 13 of the affected members, 8 of the patients were myopic, 5 were emmetropic but none were hyperopic. The occurrence of myopia is certainly more than the normal one and cannot be merely accidental or incidental.

TYPE OF GLAUCOMA

Hereditary glaucoma usually, but by no means always, appears in a chronic form. Harlan [26] reported a remarkable example in which glaucoma of the congestive type appeared in five successive generations before the age of 20. The report, however, leaves one in doubt whether the glaucoma was primary or secondary. A rapidly progressive type of glaucoma was observed by Wallenberg [27] in a pedigree of four gener-

22. Zorab, A.: Glaucoma Simplex Familialis, Tr. Ophth. Soc. U. Kingdom **52**:446-460, 1932.

23. Nettleship, E.: Glaucoma in Early Life, Roy. London Ophth. Hosp. Rep. **12**:220, 1888.

24. Somya, J. R.: Ueber erbliches Glaukom, Klin. Monatsbl. f. Augenh. **31**: 390-394, 1893.

25. Haag, C.: Das Glaukom der Jugendlichen, Klin. Monatsbl. f. Augenh. **54**:133-167, 1915.

26. Harlan, H.: A Case of Hereditary Glaucoma, J. A. M. A. **5**:285-286 (Sept. 12) 1885.

27. Wallenberg, T.: (*a*) Hydrophthalmus, Deutsche med. Wchnschr. **33**:1275, 1907; (*b*) Erbliches juveniles Glaukom, Ztschr. f. Augenh. **24**:468, 1910.

ations; all 6 members were blind as the result of glaucoma of the congestive type, and the onset occurred at the same period of life (average age, 22) in all affected members. Biro [28] reported three pedigrees of three generations. In one of these 7 members were affected with the acute inflammatory type of glaucoma between the ages of 35 and 58; in the other pedigrees there were 5 affected members in succeeding generations between the ages of 20 and 55, and all suffered from glaucoma of the inflammatory type. The type of glaucoma occurring in one family does not seem to vary, so that there is usually either a congestive or a noncongestive type of glaucoma, but both forms may alternate as shown in the table. It is possible that a local or general disposition to glaucoma is inherited and that the appearance of the disease depends on other factors, either inherited or acquired.

If it is true that congenital glaucoma (hydrophthalmia) and glaucoma of the hereditary type are essentially the same disease with the same underlying pathogenesis, then the former variety should arise commonly among the offspring of glaucomatous patients. There is as yet little evidence that such a sequence is potential. When it does occur it is of interest because it links up the two types. The younger of 4 sisters in a family reported on by Löhlein [14] had marked hydrophthalmia, and the other 3 had glaucoma which appeared at the ages of 15, 17 and 10, respectively. In a pedigree reported by Derby [29] a glaucomatous father, who had been blind for twenty-five years, had in a family of 11 children, 3 blind with glaucoma and 1 with hydrophthalmia. Berg [18] reported a pedigree of six generations in which there were 21 affected members, 19 of whom had chronic primary glaucoma and in 2 of whom hydrophthalmia developed. Scheerer [30] reported hydrophthalmia in a father and his daughter; juvenile glaucoma developed in 1 cousin at the age of 18, and another cousin had hydrophthalmia. Korte [17] reported on a family in which the father and his daughter had glaucoma simplex and hydrophthalmia developed in 2 grandchildren (1 male and the other female) by direct inheritance. The cited relation between the two groups point to an underlying genetic cause leading to a failure (more or less complete) of development of the filtration angle. If the failure is complete, congenital hydrophthalmus results; if it is incomplete, the drainage will be maintained for a variable time, until the sclerosing changes of adult life can no longer be compensated. Many hydrophthalmic eyes have been subjected to microscopic examination; in all of them abnormalities of the anterior parts of the eye

28. Biro, I.: Notes on the Heredity of Glaucoma, Ophthalmologica **98**:43-50, 1939.

29. Derby, H. C.: Three Cases of Hydrophthalmus Treated with Iridectomy, Arch. Ophth. **11**:37-40, 1882.

30. Scheerer: Demonstration klinischer Fälle, Klin. Monatsbl. f. Augenh. **89**: 820, 1932.

have been found. Examinations of excised eyes of adults with hereditary glaucoma have been made (Verhoeff[31]). Such examinations, however, are apt to be misleading for the reason that an excised eye is usually a grossly diseased eye and may be completely disorganized by previous operative intervention or by inflammatory reactions to which it has been subjected.

HEREDITY

When hydrophthalmia is transmitted it appears as a simple recessive; that is, it will not develop unless two conductors of the disease marry, when approximately 25 per cent of their children will be affected (Franceschetti[32]). An example of recessive heredity was observed by Vogt[33] in rabbits with hydrophthalmia. In one litter of rabbits, 4 were found to have marked hydrophthalmus; 2 of these were mated, and of the litter of 3, all were found to have bilateral hydrophthalmos.

Hereditary glaucoma simplex appears as a simple dominant in practically all published pedigrees reviewed by Waardenburg,[34] Franceschetti,[32] Clausen,[35] Bell[3] and others. Either parent is equally potent as a transmitter of the disease, and the parent who transmits the disease is usually affected. Hereditary glaucoma appears in otherwise healthy stock and few associated hereditary diseases or anomalies are found among the affected persons. For a long time there has been an impression that the Jewish race is more susceptible to glaucoma than the Gentile. In a review of the 27 pedigrees I have not been able to prove any racial tendency. The only pedigree of Jewish ancestry was reported by Kirkpatrick.[36] Consanguinity was reported in only 1 pedigree, and that by Plocher.[5]

My own pedigree (chart 1) was submitted to the department of genetics, Carnegie Institution of Washington, Cold Spring Harbor, Long Island, N. Y., and Dr. Barbara S. Burks made the following analysis, which I shall quote as written:

> The glaucoma pedigree has been looked over by Dr. Davenport and myself with great interest. On the whole the pedigree appears to result from a simple dominant, and clearly not from a sex-linked gene as occurs in some glaucoma pedigrees. In the case of the offspring of III-5, the question arises as to whether

31. Verhoeff, F. H., in discussion on Courtney and Hill.[4]

32. Franceschetti, A.: Missbildungen und Vererbung, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 1, p. 712.

33. Vogt, A.: Vererbter Hydrophthalmus beim Kaninchen, Klin. Monatsbl. f. Augenh. **63**:233, 1919.

34. Waardenburg, P. J.: Das menschliche Auge und seine Erbanlagen, in Bibliographia genetica, The Hague, M. Nijhoff, 1932, vol. 7, pp. 327-343.

35. Clausen, W.: Vererbungslehre und Augenheilkunde, Zentralbl. f. d. ges. Ophth. **13**:41, 1925.

36. Kirkpatrick, cited by Bell.[3]

these children are old enough to have exhibited glaucoma. It would be rare in the case of a dominant to have six normal children of an affected parent. As this is a second marriage, however, perhaps the children are younger than the other members of the 4th generation.

The apparent anticipation of age found in the 5th generation is undoubtedly due to the fact which you pointed out—that most of the offspring have not yet reached the age of onset. This would mean that those who have glaucoma would tend to be those who developed it at younger ages than typically occurs.

If one makes a count of glaucoma cases in the 4th generation and includes the offspring of III-5, the ratio of living affected to normal is approximately 50-50. If one excludes the offspring of III-5 (as being perhaps too young) the ratio of affected to normal is 7 to 2. This seems to be weighted in the direction of affected cases, but such a deviation from the 50-50 ratio might be expected if the propositus case occurs in one of the 4th generation fraternities. It is a commonplace fact, of course, that the chances of a sibship being ascertained in a study of affected patients is proportional to the number of affected offspring in the fraternity. Consequently any random set of pedigrees would be weighted with affected cases and require a correction when investigating the presence of a true 50-50 ratio.

In so far as one can judge from this pedigree, the chances that a given offspring of an affected person would be affected are 50-50. The chances that offspring of normal individuals would be affected appear to be nil.

PROGNOSIS AND TREATMENT

A disease in which the cause seems inherent, the determining mechanism of which is still unknown, naturally offers a poorer prognosis than a disturbance in which the course is transitory and its development incidental. For obvious reasons it is impossible to estimate the limit of safety in persons belonging to a family affected with hereditary glaucoma. Once the equilibrium of the pressure-regulating mechanism of the eyeball is disturbed, the tension may be raised to a certain degree without producing the common symptoms of glaucoma. Especially is this true when one considers the compensatory mechanism of the eyes of youth. It seems certain that the safe limits vary from person to person as well as in different periods of life. This may account for the lack of sustained and fixed manifestations in this disease. It seems probable that once the compensatory mechanism fails, secondary anatomic changes will result, which in turn lead to a further rise in tension and an amazingly rapid progression of all features in this disease. This was the case in most of the histories of others that were examined as well as in mine.

There seems to be no evidence offered in the literature that a beneficial outcome will result without operation. The disease cannot be favorably influenced for any length of time by the use of miotics or hypotensive drugs. Of the various operative procedures employed, iridectomy, cyclodialysis and sclerotomy have shown indifferent results, the disease advancing inexorably to complete loss of visual function.

The only permanently good results reported were in thoses cases in which one of the fistulizing operations was performed, such as the Lagrange sclerectomy, iris inclusion operations or the Elliot trephine operation. This fact bearing on the choice of operative procedures for glaucoma of the hereditary type must be borne in mind and cannot be emphasized too strongly.

In my own group of cases puncture of the anterior chamber followed in a few days by an iris inclusion operation (iridotasis) was the operation of choice when the patients were seen during the congestive stage of the disease. If the tension was very high and for any reason it was not wise to attempt fistulization immediately, it might become essential to reduce the tension as a preliminary measure by one or more punctures (paracentesis) of the anterior chamber. Sclerocorneal trepanation, following the method of Elliot, however, was preferred in all those cases in which the eyes were free from congestive signs. The best results were obtained when the operation was performed early in the disease, when glaucoma was still in the compensatory stage.

REPORTS OF CASES

The following example of hereditary glaucoma occurred in patients seen in private practice. The earlier notes on some of the affected members of the third and fourth generation of this family were made by the late Drs. Harold Gifford and James M. Patton, whose records are at my disposal. Subsequent notes were made on patients examined by myself. Statements concerning certain nonaffected persons not seen or examined can be credited as being reliable, since the information in their respective cases has been obtained from well informed and intelligent lay witnesses.

FIRST GENERATION

William S., born in England in 1799, was a farmer but had to give up his work because of visual impairment. He was completely blind for fourteen years before his death, at the age of 47. It is known that during his blindness he was led by a dog. William S., had 2 children, Joe, who never married and whose eyes were normal, and Elizabeth.

SECOND GENERATION

Elizabeth, born in England in 1834, married and emigrated to America in 1870. Her blindness came on gradually while she was still a young woman. For several years before she died she suffered from severe "neuralgic" pains in her head (absolute glaucoma ?) and was blind before her death in 1880, at the age of 46. Elizabeth S. H. had 8 children, 4 males and 4 females.

THIRD GENERATION

The children of Elizabeth S. H. are Will, Edith, Minnie, Ted, Maude, Jim, Thirza and Roland.

Will H. (no. 1), the eldest, was the only member of this generation who had not consulted an oculist. It is known, however, that he became blind while still a young man. He had 4 children, all of whom married, but no information can be obtained regarding the members of this sibship, since they became separated from their family and apparently were not in sympathy with the medical profession.

Edith H. B. (no. 2), born in 1856, is still living at the age of 84. She lost her vision early, becoming blind at the age of 19. Subsequent examinations revealed that she suffered from glaucoma, for which one eye had been enucleated. She had 7 children. Leonard and Andrew were affected with glaucoma, 1 daughter, Edith, is well, the other 4 children died of typhoid fever at the ages of 14, 9, 6 and 2 years.

Minnie H. C. (no. 3) was born in 1864. Glaucoma developed when she was 18 years old, and both eyes were operated on in Des Moines, Iowa, in 1882. Total blindness followed this operation. This patient died at the age of 26 in 1890, during childbirth. In 1891 her husband, J. A. C., married Minnie's younger sister, Maude, who also became blind due to glaucoma at the age of 24. Minnie had 5 children: Ernest, Arthur and Forest, all of whom became affected with glaucoma, and 2 daughters, Nettie and Myrtle. Nettie was the only 1 in this sibship in whom the disease did not develop.

Ted H. (no. 4) died at the age of 22 in an accident, but he also had glaucoma and was almost blind before his death. He had 2 children, Fred and Maude; glaucoma developed in Fred.

Maude H. C. (no. 5) was born in 1866. When she was 17 years old both eyes were operated on for glaucoma in Davenport, Iowa. At the age of 24 she was totally blind and later had the right eye removed at the age of 28. She married J. A. C., her sister's (Minnie) husband, when she was 25 years old. Her children—Laura, aged 43; Grace, aged 42; Rose, aged 40; Edwin, aged 31, and William, aged 30—were examined in 1936. Their eyes were still free from glaucomatous signs and symptoms.

Jim H. (no. 6), Thirza H. W. (no. 7) and Roland H. (no. 8), the youngest 3 members of this sibship of 8 in the third generation, were free from glaucoma. Their children and grandchildren, who were all examined as indicated on the pedigree (chart 1), did not acquire the disease.

FOURTH GENERATION

Leonard B. (no. 5), son of Edith H. B., single, was examined on Sept. 14, 1921, at the age of 46. He gave the history that at the age of 34 both eyes were operated on by Dr. Tiffany of Kansas City, Mo. Statements in regard to the patient's blind mother were obtained. Examination of his eyes showed evidence of bilateral iridectomy with incarceration of iris tissue in the scar. Vision in the right eye was limited to perception of light to the right side; vision in the left eye was 20/50 and was not improved with glasses. The visual fields were as shown in chart 2. Ophthalmoscopic examination showed rather deep glaucomatous excavation, more pronounced in the right eye. On retinoscopic examination a + 0.25 D. sph. was prescribed for each eye. Tension in the right eye was 12 mm. of mercury and in the left eye 12 mm. (measured with a Schiötz tonometer, as were all subsequent tonometric readings).

Andrew B. (no. 6), son of Edith H. B., married and had 2 children. He was examined on Nov. 9, 1925, at the age of 42, and gave the following history: Vision was good in each eye one year previously, but it began to fail about four months later. There had been no pain until recently. Vision in the right eye was limited

to counting fingers at 1 foot (30 cm.) and was 8/200 in the left eye with a + 0.50 D. sph. before each eye. The anterior chambers were normal in depth, perhaps very slightly deeper than usual. The cornea of the right eye was steamy and showed slight circumcorneal congestion. Tension in the right eye was 60 mm. of mercury and in the left eye 51 mm. Both optic nerves were deeply excavated. The visual fields are shown in chart 3. The use of physostigmine salicylate for the next few days did not reduce the intraocular pressure. Iridotasis was done on the right eye on November 14. Five days later a choroidal detachment was noticed in the lower one third of the fundus. On April 19, 1926, vision in the right eye was 3/200 and in the left eye 10/200. Tension in the right eye was 17 mm. of mercury and in the left eye 44 mm. Iridotasis was done on the left eye on April 28. Vision since that date has remained the same. Tension three months later was 22 mm. of mercury in the right eye and 23 mm. in the left eye.

Ernest C. (no. 12), son of Minnie H. C., aged 47, noticed a gradual failure of vision, especially in his right eye, the history of which dates back seven years.

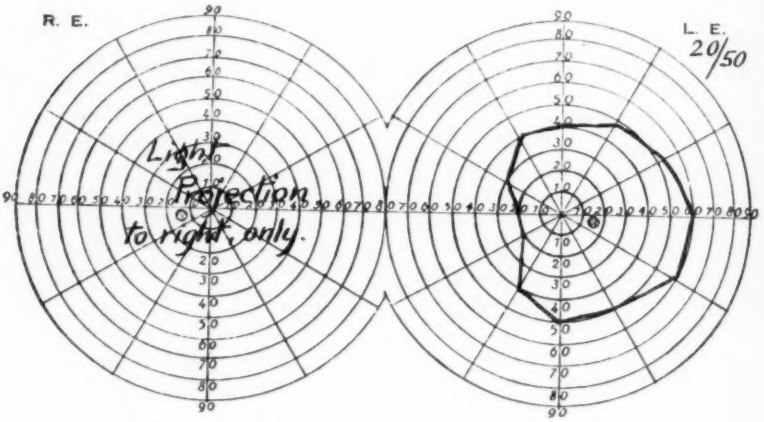

Chart 2.—Visual fields of Leonard B. on Sept. 14, 1921.

In 1923 he consulted Dr. J. B. Seeds, in Miami, Fla., who diagnosed his ocular condition as glaucoma and who, with the use of physostigmine salicylate, was able to restore his vision so that he could continue with his work as a machinist for the next three years. However, the vision in his right eye had been failing rapidly of late, and he now had difficulty with his left eye as well. Examination on Sept. 10, 1926, gave the following results: vision, perception of light in the right eye and 20/30 in the left eye; no improvement with a + 0.50 D. cyl., axis 90. The anterior chambers were normal in depth. The cornea of the right eye was slightly steamy; that of the left eye was clear. Ophthalmoscopic examination showed glaucomatous excavation in both eyes; that in the right eye was marked. Tension in the right eye was 50 mm. of mercury, in the left eye 45 mm. Physostigmine salicylate used every half hour for four hours had no effect. An epinephrine pack under the upper lid of the right eye did not reduce the tension, but a pack of physostigmine salicylate under the upper lid of the left eye reduced the intraocular pressure to 30 mm. of mercury. On September 11 iridotasis was done on both eyes. Later there was choroidal detachment in the right eye but none in the left. A little iris showed through the conjunctiva of both eyes, and there was a uni-

form filtration bleb which communicated with the anterior chamber. Tension was low to palpation. Vision in the right eye was limited to perception of light; in the left eye with a + 1.25 D. cyl., axis 150 it was 20/30. The visual fields are shown in chart 4. In 1936 the patient wrote that his eyes were just about the same as after the operation in 1926. His vision has not deteriorated in his left eye, and he reads with the aid of glasses. Ernest C., has 2 sons, aged 31 and 17, and a daughter, aged 22, who as yet are not affected with glaucoma, although none have been examined by an oculist to determine this fact.

Arthur C. (no. 13), aged 41, was examined on June 21, 1922. He complained of headaches, and because of "family eye trouble" he wanted his eyes checked. Vision in the right eye was 20/30 and in the left eye 20/20—2; with a + 0.50 D. cyl., axis 180 for the right eye vision was 20/20—3; a + 0.25 D. cyl., axis 25 for the left eye gave vision of 20/20 — 1. Ophthalmoscopic examination showed deep and rather sharp glaucomatous excavation of the nerve head in the right eye. The excavation in the left eye was shallow but suggestive of glaucoma. The

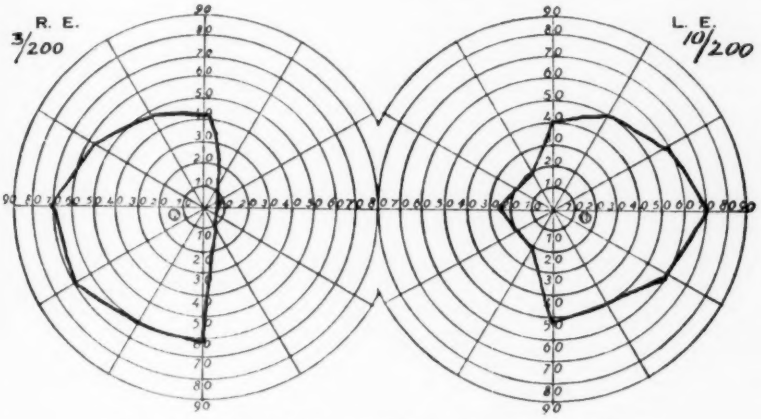

Chart 3.—Visual fields of Andrew B. on Nov. 9, 1925.

corneas measured 12 mm. in diameter. The anterior chambers were normal in depth, perhaps somewhat deeper than normal. Tension in the right eye was 32 mm. of mercury and in the left eye 31 mm. The patient was given drops of physostigmine salicylate four times daily in both eyes. He was not seen again until June 2, 1924, when he complained of pain in both eyes. Tension was 32 mm. of mercury in both eyes, although he had been using physostigmine salicylate. Vision in the right eye was 20/25 with a — 0.25 D. sph. ⊂ + 0.75 D. cyl., axis 180; in the left eye it was 20/20 with a + 0.25 D. cyl., axis 170. At this time paracentesis of both anterior chambers reduced the pressure for several days to 14 mm. of mercury. The patient returned for reexamination on Jan. 3, 1925. He had been using physostigmine salicylate in both eyes four times daily. Tension in the right eye was 35 mm. of mercury and in the left eye 17 mm. Iridotasis was done on the right eye on January 5. The patient returned on October 15, with tension of 12 mm. of mercury in the right eye and 30 mm. in the left eye. Vision with correction was 20/50 in the right eye and 20/20 in the left eye. Iridotasis was performed on the left eye on November 25. Reexamination on June 27, 1929 showed vision of 20/50 in the right eye with a — 0.75 D. sph. ⊂

— 1.00 D. cyl., axis 95; vision in the left eye with a — 1.00 D. sph. ⌒ — 2.25 D. cyl., axis 70 was 20/20. Tension in the right eye was 10 mm. of mercury and in the left eye 11 mm. Reexamination on Dec. 27, 1935 showed vision in the right eye of 20/30 with a — 0.25 D. sph. ⌒ + 0.75 D. cyl., axis 30; in the left eye vision was 20/25 — 3 with a + 1.00 D. sph. ⌒ — 2.25 D. cyl., axis 70. Tension was 15 mm. of mercury in both eyes. Corneal measurements were 12 mm. for both eyes. Filtration blebs were functioning nicely. The visual fields are shown in chart 5.

The patient returned in July 1936 with his condition unchanged. He had not been using physostigmine salicylate since the operation in 1925. Arthur C. has 9 children.

Edith, born in 1904; Ralph, in 1907; Lloyd, in 1908; Minnie, in 1910; Chalbert, in 1913; Donald, in 1916; Leslie, in 1918; Glenn, in 1922, and Grace, in 1925. Of these children, Ralph and Lloyd are affected with glaucoma in the compensatory stage. All of the children were examined.

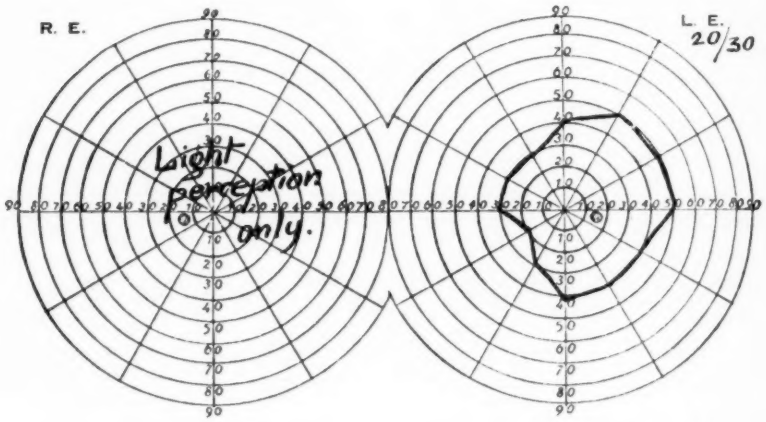

Chart 4.—Visual fields of Ernest C. on Sept. 10, 1926.

Forest C. (no. 14), son of Minnie H. C., was examined in June 1922 at the age of 37 and gave the following history: Three years before, at the age of 34 he began to experience failure of vision in both eyes. This blurring in vision came on in attacks; between the attacks his vision would return to normal. Through December 1920 his vision became notably worse, and he had some difficulty in getting about when the sun was bright. There was no pain, inflammation or headache at any time. His general health was always excellent. On Jan. 10, 1921 he was examined by the late Dr. A. C. MacLeish, of Los Angeles, and the record of the case was sent to me by Dr. Lloyd Mills, from which I wish to present the following data: When the patient was first seen the pupils were widely dilated. Tension was + 2 to fingers, the media were clear and both disks were markedly and typically glaucomatous with undercut margins. The vessels showed much angulation over the borders of the cups and pulsation; the fundi otherwise were normal. Vision in the right eye was 6/22 and in the left 6/45. Tension was 60 mm. (Schiötz) in each eye. The patient was given a 0.25 per cent solution of physostigmine salicylate to use twice a day which contracted the pupils well and brought tension to 27.5 in the right eye and 18 mm. in the left. For the next year

the history is condensed, and under miotics (physostigmine salicylate) the tension remained between 40 to 31 mm. in each eye. Combined vision remained about 6/12 + 3. On Feb. 9, 1922 an Elliot trephining operation was done on both eyes without complications. The operation was followed by the use of 1 per cent solution of pilocarpine, and vision of 4/6 resulted with the following glasses: right eye, a —1.50 D. sph. ⌒ —0.50 D. cyl., axis 180; left eye, a —1.25 D. sph. ⌒ —0.50 D. cyl., axis 180. The notes on the records stated that on March 8 tension was excellent; and the patient was going east to report to Dr. Harold Gifford. During his visit east he found out with regard to the blindness in his family.

Examination was made by the late Dr. Harold Gifford in March 1922, while the patient was traveling through Omaha and therefore could not be complete. Both eyes showed good trephine blebs with a buttonhole in the iris. Tension was normal to palpation but was not taken with a tonometer. Both eyes showed partially atrophic nerves with glaucomatous excavation. The anterior chambers were of

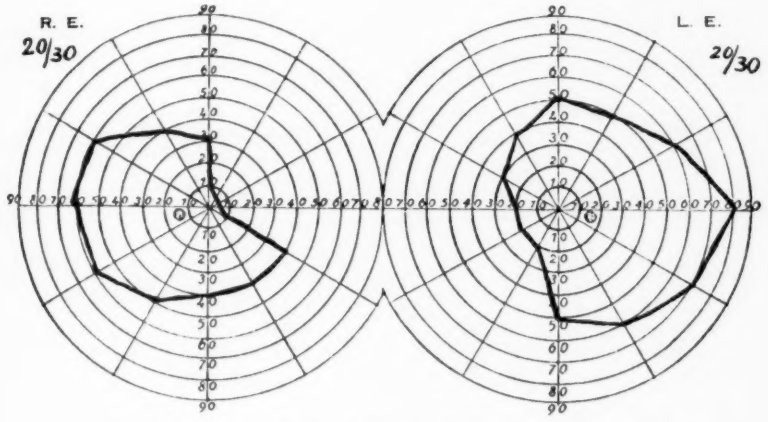

Chart 5.—Visual fields of Arthur P. C. on Dec. 27, 1935.

normal depth. The corneas measured about 12 mm. The fields were not taken. The records of Dr. MacLeish in 1927 to 1928 show a gradual rise in tension, that in 1928 being 47.5 and 44 mm. in the two eyes respectively. Finally in October 1928 the tension was found to be 60 mm. in the left eye and to be associated with much pain. The eye was enucleated (apparently the filtration bleb had closed in this eye). The last note was made on March 6, 1936. The right eye had considerable pericorneal injection with a wide pupil. The anterior chamber was clear; the optic nerve was very pale. The vision was 3/60 and the tension, 25 mm. Forest C. has 3 sons, 1 of whom was affected with glaucoma.

Myrtle B. C. (no. 16), daughter of Minnie H. C., was examined on June 21, 1922, at the age of 34, and gave the following history: She had had smallpox the previous April and had noted blurring of vision ever since. Vision in the right eye was 20/50 with a —0.50 D. sph. ⌒ +1.00 D. cyl., axis 100; vision in the left eye was 20/70 with a +0.50 D. cyl., axis 180. The anterior chambers were normal, appearing perhaps somewhat deeper than normal. Each eye had a deep sharp excavation of the optic nerve with arterial pulsation on the disk. Tension in the right eye was 60 mm. of mercury and in the left eye 65 mm.

Physostigmine salicylate administered during the next few days did not reduce the pressure. The Elliot trephine operation was performed on both eyes on July 5, with a 2 mm. blade. (A small buttonhole iridectomy was made.) Refraction on July 12 showed vision of 20/50 in the right eye with a —1.50 D. sph. ⌒ —1.00 D. cyl., axis 180; vision in the left eye was 20/50 with a —1.25 D. sph. ⌒ —0.25 D. cyl., axis 180. Tension was 15 mm. of mercury in each eye. On May 21 vision in the right eye was 20/70 with a + 1.00 D. sph. and in the left eye 20/200 with a + 1.00 D. sph. Tension in the right eye was 15 mm. of mercury and in the left eye 20 mm. The corneas measured 12 mm. The anterior chambers were of normal depth. On July 19, 1938 reexamination showed no change. The visual fields are shown in chart 6. Myrtle B. C. has 7 children: Mabel, born in 1909; Irene, in 1912; Julia, in 1915; Leona, in 1921; Margaret, in 1923; Verna, in 1927, and Lewis, in 1929. Of these, Mabel, Irene, Julia and Leona have acquired glaucoma and have been operated on.

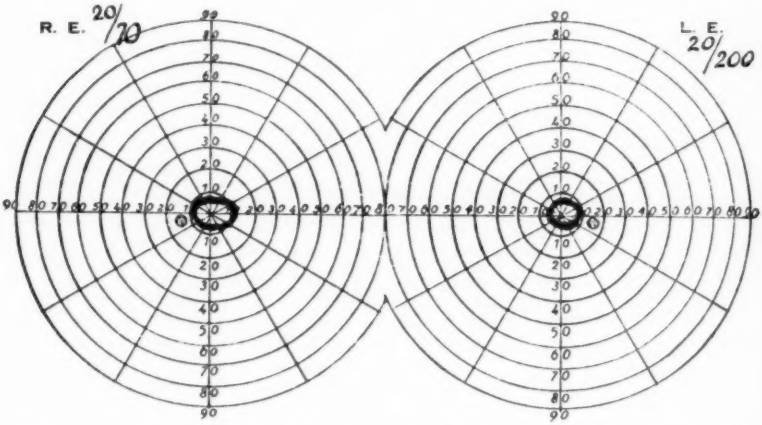

Chart 6.—Visual fields of Myrtle C. on May 21, 1936. They were the same on July 19, 1938.

Alfred H., (no. 18), son of Ted H., was examined on May 14, 1916, at the age of 49, and gave the following history. When he was 19 years old he had both eyes operated on for glaucoma by Dr. E. H. Hazen, of Davenport, Iowa. The vision in both eyes gradually failed. He could only distinguish daylight from darkness. Examination showed vision in both eyes to be limited to hand movements only. The optic nerves were atrophic and excavated. Both eyes were soft and showed beginning phthisis. The cornea in each eye was slightly opaque from interstitial opacities. The patient was unable to state with certainty what type of operation was performed. In June 1936 the patient wrote that he was 69 years old and for the past twenty years had been able to see only enough to find his way about in surroundings with which he was familiar. He never married.

FIFTH GENERATION

Ralph C. (no. 13), son of A. C., was examined for the first time on Aug. 24, 1922, at the age of 15. Vision was 20/20 in each eye with a —0.25 D. sph. The intraocular tension was 18 mm. of mercury in each eye. Ophthalmoscopic examina-

tion gave negative results. There was no excavation of the nerve head. Reexamination on June 2, 1924 showed the corneas to measure slightly over 12 mm. in diameter. The anterior chambers were normal in depth. Refraction showed vision in the right eye of 20/20 with a — 0.25 D. sph. ⌒ — 0.25 cyl., axis 180; vision in the left eye was 20/20 with a — 0.25 D. sph. Tension was 23 mm. of mercury in each eye. Reexamination on Aug. 4, 1936, at the age of 29, showed vision of 20/20 in each eye with the foregoing correction. The anterior chambers were of normal depth. The iris stroma was thin. The fundi showed deep physiologic (?) cupping. The vessels were pushed toward the nasal side; the origin of blood vessels (dipping over the edge of the cups in both eyes) was not seen. The fields were normal to form and to color. On a tangent screen at 1 meter, with a 3 mm. white test object, there was a scotoma extending up and out from the right blindspot. The left eye was normal. Tension in the right eye was 26 mm. of mercury and in the left eye 24 mm. A diagnosis was made of early glaucoma but still in the compensatory stage (?). Use of a 1 per cent solution of pilocarpine was prescribed.

Lloyd C. (no. 14), son of A. C., was first examined on Aug. 24, 1922, at the age of 13. Vision in the right eye was 20/20 with a — 0.50 D. sph. ⌒ — 0.25 D. cyl., axis 180; in the left eye it was 20/20 with a — 0.25 D. cyl., axis 180. The corneas measured 12.5 mm. The anterior chambers were normal in depth. Reexamination on Dec. 27, 1935 showed vision in the right eye of 20/20 with a — 0.50 D. sph. ⌒ — 1.25 D. cyl., axis 85; vision in the left eye was 20/20 with a —1.00 D. sph. Tension in the right eye was 26 mm. of mercury and in the left eye 25 mm. The fundi and fields were normal. A 1 per cent solution of pilocarpine was instilled into each eye three times a day, which reduced the tension to 20 mm. of mercury in both eyes. Reexamination on June 11, 1936 showed vision of 20/20 with correction. The patient had been using pilocarpine morning and evening. Tension in the right eye was 29 mm. of mercury and in the left eye 26 mm. Reexamination on Feb. 2, 1940 showed vision in the right eye of 20/20 with a — 0.50 D. sph. ⌒ — 2.25 D. cyl., axis 80; vision in the left eye was 20/20 with a — 1.25 D. sph. ⌒ — 0.50 D. cyl., axis 105. The corneas measured 12.5 mm. The depth of the anterior chamber was 3.5 mm. in each eye. The fields and fundi were normal. Tension in the right eye was 28 mm. of mercury and in the left eye 26 mm. (with the use of pilocarpine morning and evening). A diagnosis was made of early glaucoma in the compensated stage. The patient was instructed to continue to use the pilocarpine morning and evening and to return in three months.

Lester C. (no. 23), son of Forest C., was seen by Dr. A. C. MacLeish (now deceased). The following data were sent to me by Dr. Lloyd Mills, of Los Angeles: Examination of the patient was made on Aug. 29, 1928, at the age of 20. His complaint was blurred vision in the right eye, which had been noted for about three years. His combined vision was 6/12. The pupils were dilated, both disks were markedly glaucomatous with deep cups, and there was strong angulation of the vessels. Tension (Schiötz) was 55 mm. in each eye; it improved to 43 and 39 mm. respectively by the use of a 1 per cent solution of pilocarpine three times a day. Corrected vision was: right eye, 6/5 with a — 1.50 D. sph. ⌒ — 0.62 D. cyl., axis 90; left eye, 6/5 with a — 1.00 D. sph. ⌒ — 1.00 D. cyl., axis 90. In spite of miotics, the tension remained up, and on October 18 an Elliot trephine was done in both eyes without complications. In 1929, two and one-half hours after the use of drops, tension was 22.5 mm. in the right eye and 40 mm. in the left eye. No mention was made of the visual fields. Vision in 1930, 1932 and 1934 remained at 6/5 in each eye. In August 1935 tension in the right eye was 22 mm. and

in the left eye 27.5 mm., and vision was still 6/5 with a compound myopic correction. There has been no record since.

In a recent communication from the patient it was ascertained that he has had his glasses changed on several occasions and each time with good visual results.

Mrs. Mabel J. (no. 27), daughter of M. B. C., was examined for the first time on June 5, 1936, at the age of 26. She noticed a change in her vision for distance for the past six months. She had never worn glasses. Vision in the right eye was 20/20 — 4 with a — 1.50 D. sph. ⌒ — 0.75 D. cyl., axis 95; in the left eye it was 20/20 — 3 with a — 0.75 D. sph. Ophthalmoscopic examination of the right eye showed the optic disk deeply excavated, the excavation extending to the scleral ring nasally, below and temporally. The vessels were out of sight. In the left eye the glaucomatous cupping was not quite as deep as in the right. Excavation of the nerve head measured about 3 diopters in the right side and about 2 diopters in the left. Corneal measurements were 12 mm. The depth of the anterior chamber

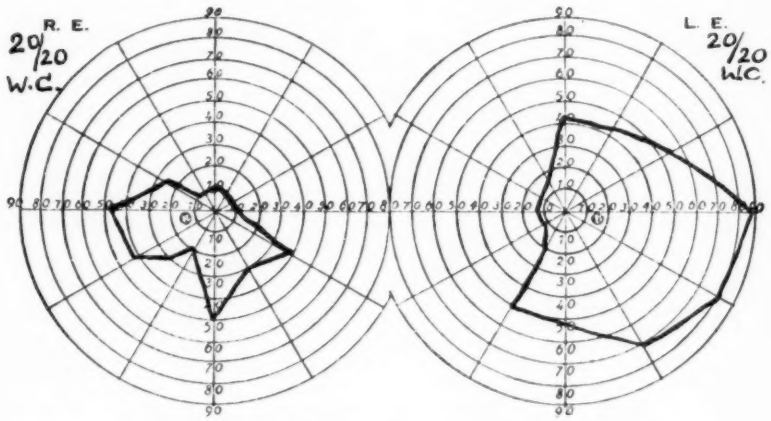

Chart 7.—Visual fields of Mrs. M. J. on June 5, 1936.

was 4 mm. in the right eye and 3.5 mm. in the left. Intraocular tension in both eyes was 56 mm. of mercury. It was reduced to 34 mm. in two hours in both eyes after instillation of physostigmine salicylate every ten minutes, six times. The visual fields are shown in chart 7. Trepanation was done on the right eye, on June 11, with a 2 mm. blade, and on the left eye on June 22, with a 2 mm. blade. Reexamination on July 28 showed the eyes white and quiet. Tension was low to palpation. Vision in the right eye was 20/25 with a — 1.50 D. sph. ⌒ — 1.25 D. cyl., axis 165; in the left eye it was 20/20 — 3 with a — 0.75 D. sph. General physical examination gave entirely negative results. There was nothing to indicate any endocrine or vasomotor instability as a possible etiologic factor in glaucoma. Reexamination on Feb. 3, 1940 showed vision in the right eye of 20/25 with a — 0.37 D. cyl., axis 150, and in the left eye of 20/20 — with a — 0.75 D. sph. ⌒ —0.50 D. cyl., axis 45. There was a good filtration bleb in both eyes. Ophthalmoscopic examination showed little evidence of glaucomatous cupping, certainly not nearly as marked as it was on June 11, 1936. Tension in the right eye was 12 mm. of mercury and in the left eye 14 mm. The visual fields showed slight improvement over those of June 5, 1936. Mrs. M. J. has 3 children, aged 3 to 8 years. Their eyes were examined and are still normal.

Mrs. Irene C. (no. 28), daughter of M. B. C., was examined on May 21, 1936, at the age of 24. The patient stated that she started to wear glasses when 16 years old and for the past three years had been wearing a —3.50 D. sph. for each eye. Vision was 20/20 in the right eye with —4.75 D. sph. ⌒ —0.25 D. cyl., axis 90; it was 20/20 in the left eye with a — 5.00 D. sph. The corneas measured 12 mm., the anterior chambers were 3.5 mm. in depth. Ophthalmoscopic examination showed early glaucomatous excavation of the nerve head in both eyes. The visual fields showed contraction for form and color in the lower nasal quadrant. The intraocular tension was 26 mm. of mercury in both eyes. The patient was given a 1 per cent solution of pilocarpine to be used three times daily. Reexamination on August 20 showed vision in the right eye of 20/20 — 4 and in the left eye of 20/20 with glasses. Tension in the right eye was 35 mm. of mercury and in the left eye 41 mm. under the effect of pilocarpine. Trepanation was performed on August 21 on both eyes, a 2 mm. blade being used. Reexamination on Feb. 3, 1940 showed vision in the right eye of 20/20 with a —5.50 D. sph.; in the left eye it was 20/20 with a —4.25 D. sph. ⌒ —1.25 D. cyl., axis 5. Tension in the right eye was 15 mm. of mercury and in the left eye 14 mm. The fields were normal for form. The patient has 3 children; all were examined and are free from glaucoma at the ages of 1, 3 and 5 years.

Mrs. Julia G. (no. 29), daughter of M. B. C., was examined on Sept. 9, 1936, at the age of 20. She had had no particular difficulty with her eyes but for the past few months had noticed some blurring in her vision. Vision was 20/20 in both eyes. The corneas measured 11.5 mm. The depth of the anterior chamber was 3.8 mm. in the right eye and 4 mm. in the left eye. Ophthalmoscopic examination showed early glaucomatous excavation, more marked in the right eye. The visual fields were normal for form but showed contraction for colors, particularly in the lower nasal quadrant. Intraocular tension in the right eye was 48 mm. of mercury and in the left eye 40 mm. The patient was given a 1 per cent solution of pilocarpine every hour for the next few days, and the pressure was reduced to 34 mm. of mercury in each eye. On September 12 a trephine operation was done on both eyes with a 2 mm. blade. On Jan. 30, 1937 vision in the right eye was 20/20 with a —1.00 D. cyl., axis 80; in the left eye it was 20/20 with a —0.50 D. sph. ⌒ —1.00 D. cyl., axis 90. There was a good filtration bleb. On Oct. 21, 1939 vision in the right eye was 20/20 with a +0.37 D. cyl., axis 60; in the left eye it was 20/20 with a +0.25 D. cyl., axis 75. The visual fields were normal. Ophthalmoscopic examination showed little excavation. The intraocular tension was 16 mm. of mercury in both eyes. The patient is married but has no children.

Leona C. (no. 30), daughter of M. B. C., was examined on May 21, 1936, at the age of 15 years. She was tested for glasses one year before and was wearing a —0.50 D. sph. before the right eye and a —1.00 D. sph. before the left eye, which gave her vision of 20/30 in the right eye and vision of 20/40 in the left eye. She stated that her vision had been getting worse during the past six months. Ophthalmoscopic examination showed a small posterior capsular opacity on the posterior pole of the lens of the right eye. The nerve head was excavated and showed the characteristic vessel dipping of early glaucoma. In the left eye the lens was clear, and there was a characteristic glaucomatous excavation present, with vessels dipping over the periphery of the nerve head in its entire circumference. Vision in the right eye was 20/30 +2 with a —1.50 D. sph.; in the left eye it was 20/20 —2 with a —2.00 D. sph. ⌒ —0.75 D. cyl., axis 105. The cornea measured 12 mm. in diameter in each eye, and the anterior chambers were 4 mm. in depth. Intraocular

tension measured 30 mm. of mercury in each eye. The fields were normal for form but slightly contracted for colors. The patient was given physostigmine salicylate, 1 drop into each eye three times daily. Reexamination on July 28, 1936 showed the intraocular pressure to be 35 mm. of mercury under the influence of the drug. Bilateral trephining according to Elliot was done on July 29, a 2 mm. blade being used. Reexamination on Nov. 29, 1938 showed vision of 20/30 + 2 in the right eye with a —2.00 D. sph., in the left eye it was 20/20 — 1 with a —2.00 D. sph. ⌒ —0.50 D. cyl., axis 115. The peripheral field was normal, and the intraocular tension in the right eye was 17 mm. of mercury and in the left eye 14 mm.

SIXTH GENERATION

The members of the sixth generation, as indicated in the pedigree chart, were examined, but as yet are too young to have the disease. They were all free of symptoms and their eyes were apparently normal when last seen.

SUMMARY

Hereditary primary glaucoma appears to result from a simple dominant and clearly not from a sex-linked gene.

So far as one may judge from this pedigree, the chance that an offspring of a glaucomatous person would be similarly affected is in a ratio of about 50:50. The chance that an offspring of normal persons would be affected appears to be negligible. Consanguinity did not occur in this pedigree.

The age at which the rise of intraocular tension appears varies in different families. Examples can be found in which the onset of the disease occurs in early youth (juvenile glaucoma) in adolescence and in adult life. Since it is not known what proportion of cases of primary glaucoma are free from genetic potentialities, interest should be aroused in the hereditary tendency of the disease in all cases, but most especially so when the young adult is affected.

The onset of the disease may be so gradual that its date can seldom be fixed with any degree of accuracy. The reduced age of onset in the younger generation must not be taken as proof of a general tendency to anticipation of the disease.

The liability to transmission by the two sexes is equal.

Hereditary glaucoma usually, but by no means always, appears in a chronic form. It is possible that a local or general disposition to glaucoma is inherited and the appearance of the disease depends on other factors, either inherited or acquired.

The horizontal corneal measurements showed the cornea to be between 11.6 and 12.5 mm. in size.

The anterior chambers are of normal depth, averaging about 3.5 mm.

The incidence of myopia in hereditary glaucoma is sufficiently great to suggest that the hypertension had been influential in producing this state of refractive abnormality.

It is impossible to estimate the time limit of safety in persons belonging to a family affected with hereditary glaucoma. Once the equilibrium of the pressure-regulating mechanism of the eyeball is disturbed, the tension may be elevated to a certain degree without producing the common symptoms of glaucoma. Especially is this true when one considers the compensatory mechanism of the eyes of youth. When, however, this compensatory mechanism fails, secondary anatomic changes result, which in turn lead to a further rise in tension and to an amazingly rapid progression of all the features of this disease.

There is no evidence that a beneficial outcome will result without operation. The only permanently good results were obtained in persons in whom one of the fistulizing operations was performed, such as the Lagrange sclerectomy, iris inclusion operations or trepanation by the method of Elliot.

The best results were obtained when the operation was performed early in the disease, when glaucoma was still in the compensatory stage, that is to say, before irreversible secondary anatomic and pathologic changes had occurred.

COLORED CHARTS AS A SUPPLEMENTARY TEST FOR MACULAR VISION

PRELIMINARY REPORT

SAM ENGEL, M.D.
SAN FRANCISCO

Every ophthalmologist has experienced the difficulty encountered in the determiation of a minute central scotoma. This has repeatedly been stated by authors known for their work in perimetry and has been personally confirmed by my colleagues. A patient may say that he has difficulty in reading or that he tires easily on doing close work, or he may state positively that he has an impairment of his sight. Examination shows his visual acuity to be normal, and although one cannot make out a defect of the central field with the usual methods, his trouble definitely suggests the presence of a central scotoma. As Traquair [1] wrote: "Difficulty may arise in connection with the patient. A few patients although they have sufficient central vision cannot be induced to fix correctly, but persist in following a test-object with the eye." The value of Haitz's stereoscopic charts, Lloyd's stereocampimeter and Walker's macular selector is to be acknowledged, but they do not overcome all difficulties because of the required intelligence on the part of the patient and the necessary careful adjustment. Furthermore, heterotropia or defective vision of the other eye prohibit their use.

The idea of the chart described here was to enforce macular fixation by decreasing the visual angle of the test object and simultaneously to present these test objects in color on a gray background. If the patient was able to recognize such a figure while its edges were subtending an angle of 1 minute, this could be accomplished only by macular vision and his reading could be taken as an evaluation of his central color sense. Hess's numbers [2] were used as figures and were painted in the primary colors, red, green and blue, of Hering's color circle.[3] One minute was indeed the average minimum separable for a group of nor-

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From the Department of Ophthalmology, Stanford University School of Medicine.

1. Traquair, H. M.: An Introduction to Clinical Perimetry, ed. 3, St. Louis, C. V. Mosby Company, 1938, p. 43.

2. Von Hess, C.: Internationale Sehproben unter Verwendung der Landolt'schen Rings hergestellt, Munich, J. F. Bergmann, 1926.

3. Hering, E.: Grundzüge der Lehre vom Lichtsinn, Leipzig, Wilhelm Engelmann, 1905.

mal persons tested with the chart. This therefore represents the normal central color sense, marked as 10/10 or 20/20; the larger figures were graded according to the visual angle. Although the black and white chart gives a normal reading, one may find a diminished acuity with the color chart, 8/10, e. g., indicating an impaired macular function. Such conditions might readily escape notice during a busy office hour. Quoting once more from Traquair [4]: "The presense, however, of 6/9 or 6/6 with a normal field periphery does not necessarily indicate a normal condition of the central field, for normal central vision is sufficiently acute to undergo considerable impairment before an obvious reduction is demonstrable by a test such as Snellen's types."

Of course, the information obtained by the color chart is restricted to a very narrow, although most important, central district. A normal finding does not exclude a paracentral scotoma; a diminished grade of color sense does not inform one as to the shape or extent of the present depression of the central field. The purpose of this examination, which requires only a few minutes, after refraction has been determined, is only to ascertain whether or not the central color sense is normal. An impairment demands thorough campimetric study of the central field and likewise thorough ophthalmoscopic examination of the macular region after dilatation of the pupil. Nevertheless, even if one is not able to prove the existence of a central scotoma in such a patient, the diminished central color sense indicates a lesion of the macular area or of the maculopapillary bundle, rare cases such as hereditary color blindness or brownish discoloration of the lens being excluded. A routine examination of the central field is made superfluous, which not only means a saving of time but avoids the superficiality often met in a routine examination which is performed without a reasonable indication.

In two other respects the color chart seems to be advantageous. It is a simple method for measuring and comparing the grade of disturbance in the course of a disease and may be valuable in therapeutic considerations. On the other hand, it gives a better impression regarding a patient's trouble, which often does not seem to correspond to the visual acuity found with the black and white chart, for the black and white chart gives optimal conditions not always met during a patient's work or daily life.

Short reports of some cases follow for illustration:

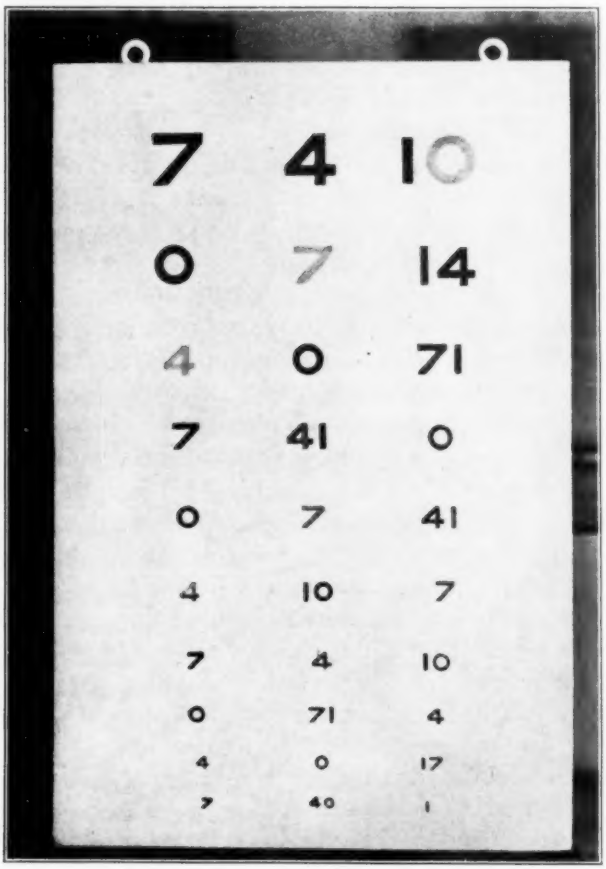
Case 1.—Mrs. J. K., aged 46, felt tired after reading for a short time; her glasses had been changed twice previously without improving her difficulties.

Vision in the right eye with a + 0.75 sph. was 20/20 (partially); with a + 2.0 sph. it was 0.40/0.50. Vision in the left eye with a + 1.0 sph. ⊃ + 0.5 cyl., axis 0 was 20/20; with a + 2.25 sph. ⊃ + 0.5 cyl., axis 0 it was 0.40/0.40. Minute

4. Traquair,[1] p. 15.

colored marks were easily recognized with either eye; there was no difference of color between central and paracentral fixation. It is understandable that vision of 20/20 (partially) was regarded as satisfying by previous examiners. Test with the new color chart gave the following results: for the right eye, red 8/10 and blue 8/10; for the left eye, red 9/10 and blue 8/10.

Both disks were normal. The reflex of the arteries was slightly increased. A triangular district of cloudy appearance and grayish red color was seen in



Colored chart, taken with a panchromatic film. Blue appears as black, red as dark gray and green as light gray.

the right macula. The left macular reflex was indistinct and surrounded by fine pigmentations.

A diagnosis was made of macular degeneration in an early stage, more pronounced on the right.

The color chart revealed impairment of the central color sense which escaped notice when other methods were employed.

CASE 2.—Mr. M. K., aged 62, always felt fatigued when reading. He had an attack of coronary thrombosis several weeks before the present examination.

When I saw him the first time the vision of the right eye was diminished to 20/40 and a central scotoma was present for white and for colors. The retinal vessels showed signs of atherosclerosis, but both disks and maculas were normal. A diagnosis of retrobulbar vascular disturbance of the right optic nerve was made.

The vision of the right eye improved slowly in the course of the following weeks, and the finding was: 20/20 (partially) with a —0.25 cyl., axis 90; vision in the left eye was 20/20 with a —0.5 cyl., axis 90. A central scotoma for white and for colors was no longer demonstrable by the test used previously, and the patient noticed only a slight difference in the color when seen with either eye but was not able to say which color he would regard as normal.

The color chart gave the following results: for the right eye, red 6/10, green 6/10 and blue 7/10; for the left eye, red 9/10, green 9/10 and blue 9/10.

The color sense of the right eye is still impaired. In this stage it was not possible to prove the change otherwise. Perception for red and green are more involved than for blue, indicating a lesion of the nerve fibers.

CASE 3.—Mr. F. R., aged 39, an instrument maker, noticed difficulty in working for one month; he saw a "granular lump" before the left eye. He was under medical care for renal disease (albumin ++; increased nonprotein nitrogen of the blood; blood pressure, 230 systolic and 150 diastolic).

Vision in the right eye was 20/20 with a +0.75 sph.; in the left eye it was 20/25, with a central scotoma for blue.

There were edema of both disks with capillary hyperemia and flame-shaped hemorrhages. Yellowish white glistening lesions, arranged in a stellate figure, were seen nasally to the left macula, which appeared blurred.

A diagnosis of neuroretinitis albuminurica was made.

Tests with the color chart revealed: for the right eye, red 9/10, green 8/10 and blue 6/10; for the left eye, red 6/10, green 6/10 and blue 5/10.

The color sense is more impaired for blue than for red and green, the cause being a nutritional disturbance of the retina. The left eye shows an early and pronounced change for blue and still has a normal visual acuity with the black and white chart.

CASE 4.—Mr. I. K., aged 60, had glaucoma simplex. Vision in the right eye was 20/20. The peripheral field for white (a 5 and a 1 mm. test object at 300 mm.) and red were normal; the blindspot was slightly enlarged. Vision in the left eye was limited to perception of hand movements; the temporal field was small. The right disk was of normal color and not excavated. Tension was well controlled by miotics.

Tests with the color chart (for the right eye, red 8/10, green 8/10 and blue 10/10) revealed a slight impairment for red and green (lesion of the nerve fibers), which will be watched in the course of treatment.

CASE 5.—Mr. W. P. was sent by his physician whom he had consulted because of headache. The ocular findings were normal. Test with the color chart revealed red 1/10, green 7/10 and blue 10/10 for either eye. Stilling's charts confirmed a red-green color blindness.

Before applying the colored numbers, I had a chart made of Landolt's rings in gray on a white ground, the gap of the ring being filled with a colored square. The patient had to locate the change in the ring and

to determine the color of the square. From a scientific standpoint, I would have preferred the rings to figures, because the recognition is dependent only on the angle of vision and the form sense plays no part. Practical application showed the figures superior to the rings, because the examination of normal persons with the rings gave marked differences, probably caused by individual differences of the gift of observation and of fatigue. One has to keep in mind that all visual tests are empiric tests, and experience with the colored numbers was satisfactory, giving practically the same response in normal persons.

The discussion about applying color in perimetry may be brought forward in this respect. I regard color perimetry as a valuable clinical test which one should not omit, as I stated in a former paper.[5] It is known that the color sense is impaired in a specific sequence regarding the individual colors, red and green being first affected in lesions of the nerve fibers, while a change for blue is demonstrable in beginning nutritional disturbances of the retina (as indicated by the preceding case histories). Peter[6] proved an indistinct Bjerrum scotoma for green to be the first change of the field in glaucoma, which "the author has been able to elicit when the minutest test object for white failed to disclose any loss of sensitivity." In hypophysial tumors I found it valuable to compare the colors in the nasal and temporal parts of the field; the patient sometimes stated an obscuration of the color in the temporal field when I was not able to prove an impairment by quantitative perimetry. Lately Peter[7] again emphasized the importance of color perimetry in saying: "One is missing too much if one fails to use colors in those instances in which experience teaches that color as well as form should be used."

In a recent paper Comberg[8] published a chart with black figures on a gray background to test patients with slight opacities of the refracting media, as for instance a beginning cataract or a delicate secondary cataract. This test increases the requirements met by the patient. This was accomplished before in a similar way when the illumination of the chart was reduced by a rheostat or by a diaphragm. The color chart combines an increase of the difficulties with a specific functional test of the macula and may besides be used with red-green glasses to prove monocular simulation.

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5. Engel, S.: Störungen des Gesichtsfeldes, Fortschr. d. Neurol., Psychiat. **3**:388 (Sept.) 1931.

6. Peter, L. C.: The Principles and Practice of Perimetry, ed. 4, Philadelphia, Lea & Febiger, 1938, p. 188.

7. Peter, L. C., in discussion on Traquair, H. M.: Clinical Detection of Early Changes in the Visual Field, Arch. Ophth. **22**:947 (Dec.) 1939.

8. Comberg, W.: Sehproben mit gemindertem Kontrast zwischen Sehzeichen und Grund als weiteres Mittel zur Erfassung der Leistung des Auges, Klin. Monatsbl. f. Augenh. **103**:466 (Oct.) 1939.

SUMMARY

The examination of a patient with the black and white chart is supplemented by a test with colored figures on a gray ground. This test can be performed in a few minutes and is particularly indicated when the visual acuity is approximately normal but the patient's statements suggest a slight impairment of macular function.

The test makes it possible to compare the impairment during the different stages of the disease. More exact information about macular vision leads to a better understanding of the patient's difficulties.

The color chart may be used with red-green glasses to prove monocular simulation.

This colored chart is available at Trainer and Parsons, opticians, San Francisco, and is also planned as a chart for projection.

TENDON TRANSPLANTATION FOR PARALYSIS OF THE EXTERNAL RECTUS MUSCLE

A FURTHER REPORT

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CHICAGO

The following report was stimulated by a recent statement of Bielschowsky.[1] "By transplantation," he stated, "one may obtain a decrease in the deviation and even a certain amount of abversion, but the patient will not be able to see objects in front of him single with the normal position of the head, and he will not abandon the habit of anomalous rotation of the head toward the paralyzed side." This statement from the world's greatest authority on the extraocular muscles would certainly tend to discourage the ophthalmologist who has never performed the operation from attempting it. It is for this reason that it seemed useful to review the results in a series of operations performed since my report in 1929.[2] At that time previously reported cases were discussed, including those of Hummelsheim, O'Connor, Peter, Posey, Franklin and Cordes and others. Two personal cases of paralysis of the external rectus muscle were reported in which this method of operation was used with good results.

Since 1929 few reports on the method have appeared in the literature. O'Connor[3] in 1930 and 1935 discussed his experiences with various modifications of the Hummelsheim procedure and gave his reasons for preferring in cases of complete paralysis use of the inner two thirds of the vertical rectus muscles. He has performed approximately thirty transplantations by all methods and expressed the belief that the results are best when the latter procedure is employed. Thirteen operations for paralysis of the external rectus muscle and four for paralysis of other muscles are reported in which his present technic was employed. In most cases O'Connor seems to perform central tenotomy of the internal rectus muscle six weeks before transplantation of the tendon, at which time lateral tenotomy may be added if necessary. In some cases of

From the Department of Ophthalmology, Northwestern University Medical School.

1. Bielschowsky, A.: Am. J. Ophth. **22**:723, 1939.

2. Gifford, S. R.: Muscle Transplantation for Paralytic Strabismus, Arch. Ophth. **2**:651 (Dec.) 1929; Am. J. Ophth. **18**:813, 1935.

3. O'Connor, R.: Am. J. Ophth. **18**:813, 1935.

partial paralysis adequate correction was accomplished by transplantation alone, without any interference with the internal rectus muscle. He affirmed the definite indication for tendon transplantation in any case of complete paralysis, advising that for treatment of the congenital form it be performed at the age of 3 and for the treatment of the acquired form, before secondary contracture has occurred. Green [4] reported a case of congenital paralysis of the external rectus muscle with Duane's syndrome in which practically the same operative method was used as that employed in my cases with partial tenotomy of the internal rectus muscle. Eighteen to 20 degrees of abduction resulted from the operation, with complete relief of head tilting in the patient, a child of 22 months. Recently Green [5] reported a second case in which the deviation was corrected and good abduction obtained. Apple [6] reported 2 cases of Duane's syndrome in which division of abnormal bands beneath the internal rectus muscle produced a marked improvement. No transplantations were performed. Vogt [7] reported two excellent cosmetic results obtained by tenotomy of the opponent and a special method of applying tension sutures to hold the eye in an overcorrected position during healing. While his two results were good, I have seen a similar use of tension sutures after tenotomy in such cases fail to accomplish a permanent improvement. Wiener [8] in 1936 described a new procedure. The external rectus muscle was cut off 15 mm. from its insertion, and the portion remaining attached to the globe was split into two halves. The upper half was sutured to the tendon of the intact superior rectus muscle and the lower half to the tendon of the inferior rectus muscle. No individual case reports are given in his original article, but he stated that the results were good. Recently [9] he reported a case in which the eyes were made straight and considerable abduction was obtained. Smith [10] has performed the original Hummelsheim transplantation operation in 4 cases, 3 of congenital and 1 of acquired paralysis, with good results, abduction being 30 degrees in some cases. Eber [11] reported 2 cases in which abduction of 30 and 40 degrees was obtained by this method.

4. Green, J.: Am. J. Ophth. **18**:58, 1935.

5. Green, J.: Am. J. Ophth. **22**:1286, 1939.

6. Apple, C.: Am. J. Ophth. **22**:169, 1939.

7. Vogt, A.: Klin. Monatsbl. f. Augenh. **103**:296, 1939.

8. Wiener, M.: Surg., Gynec. & Obst. **62**:487, 1936.

9. Wiener, M., in discussion on Green.[5]

10. Smith, E. T.: Australian & New Zealand J. Surg. **5**:219, 1936; abstracted, Zentralbl. f. d. ges. Ophth. **36**:206, 1936.

11. Eber, C., in discussion on Green.[5]

Since my last report I have used the method of tendon transplantation in 11 cases of paralysis of the external rectus muscle, of which records are available. At least 3 other cases in which operation was done are not included because of insufficient records, which, with the 2 cases previously reported, makes 16 cases in which the operation was done. The technic differed little from that described in 1929. It has been found safe to employ half of the vertical rectus muscles instead of one third without interfering with the balance of the vertical muscles. The outer halves are still employed, in spite of O'Connor's reasoning, because of a fear that adhesions might develop where the outer half of the tendon passed beneath the inner half and because the amount of effect obtained by the original method seemed adequate.

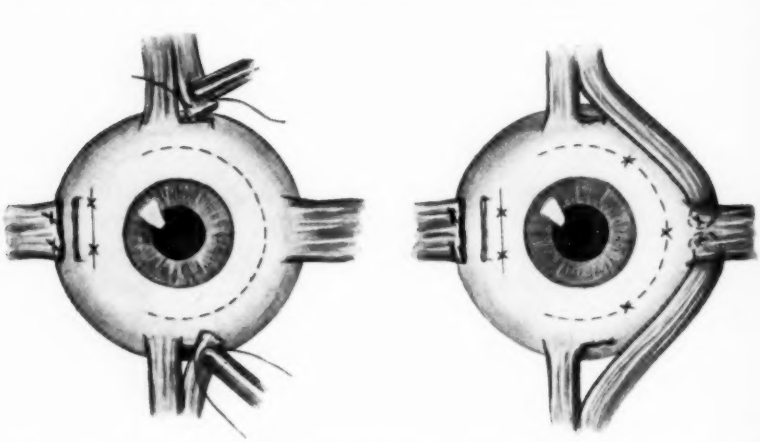

Fig. 1.—Technic of tendon transplantation with recession of internal rectus muscle.

Twenty day chromic catgut sutures (0000) on atraumatic needles are employed instead of silk. Recession of the internal rectus muscle has been preferred to complete tenotomy in some cases, especially those in which secondary contracture was not marked. The operation is performed, as a rule, with the patient under general anesthesia, to avoid infiltration in the region of four muscles (fig. 1). The data and results in the 11 cases in which this operation was performed are summarized in the accompanying table. All patients except those in cases 2 and 5 have been observed for more than one year and most for three to ten years after operation. In 8 cases the final result may be considered perfectly satisfactory, no deviation being present on cover test and abduction varying from 15 to 40 degrees as measured on the perimeter. In only 1 case (case 6) was any vertical deviation or imbalance produced by the

Results of Operation in Eleven Cases

Case	Age, Yr.	Sex	Type of Paralysis	Deviation Before Operation	Abduction Before Operation	Operation	Deviation After Operation	Abduction After Operation	Additional Notes
1	33	♂	Traumatic (skull fracture)	30°	To midline	Transplantation with recession of left internal rectus muscle	None on cover test; Maddox rod test, exophoria, 2°	22° without diplopia, 30° with right eye covered	Marked overeffect for first 2 mo.
2	12	♀	Acquired (cause unknown)	15°	15°	Transplantation alone; recession had been done elsewhere	None; Maddox rod test, esophoria, ½° for distance and none for near vision	15°	Had marked secondary deviation of right eye on looking to midline; now can turn both eyes to the left 7½° without deviation and holds head straight
3	12	♀	Congenital; slight narrowing of fissure on adduction	25° in 10° up	5°	Transplantation and recession of internal rectus muscle	Divergence, 10°	23°	Insufficient effect from first operation; later tenotomy of internal rectus muscle and advancement of external rectus muscle produced overeffect
4	41	♂	Acquired (syphilis)	16°	To midline	Recession of left internal rectus muscle and transplantation	None on cover test; Maddox rod test, esophoria, 1½°	35°-40°	
5	22	♀	Congenital with refraction syndrome	20°	To midline	Recession of internal rectus muscle of 5 mm. and transplantation; later advancement of internal rectus muscle	None on cover test	20°	After first operation had 15° of exotropia; internal rectus muscle exposed 3 mo. later; found 11½ mm. back of limbus and advanced 9 mm.; result good, with 45° of adduction and 20° of abduction
6	44	♂	Acquired (skull fracture, bilateral)	R.E., 30° L.E., 50°	R.E. to 20° from midline; L.E. remains at 40° convergence	Bilateral tenotomy of internal rectus muscle and transplantation (20 operations)	R.E., none; L.E.,15°; convergence	R.E., to midline; L.E., 10°-15°	Marked contracture of both internal rectus muscles; second operation on left eye performed 2 yr. later; recession of left internal rectus muscle and advancement of external rectus muscle with tension sutures with result as stated
7	18	♀	Congenital	25°	5°	Transplantation with tenotomy of internal rectus muscle	None on cover test; Maddox rod test, exophoria, 3°; left hypophoria, 3° for distance	40°	Adduction limited to 15° but no complaint
8	14	♂	Traumatic; bilateral	About 50°	Neither eye could be brought to midline	Bilateral recession of internal rectus muscle and transplantation	Straight on cover test	R.E., to midline; L.E., 15°	Extreme contracture of both int. rect. muscles was present
9	7	♀	Congenital with retraction syndrome	25°	To midline	Tenotomy of left internal rectus muscle and transplantation	Straight on cover test	Can abduct 15° when right eye is covered; with both eyes open, only 5°	Observed 13 yr. after operation; secondary deviation of right eye on looking to left just 5°
10	46	♀	Acquired following removal of orbital tumor	15°	To midline	Recession of left internal rectus muscle and transplantation	None on cover test; Maddox rod test, esophoria, 5°	17°	Adduction limited to 30°
11	3½	♀	Congenital (mother also has abducens paresis of left abducens nerve)	15°	To midline	Guarded tenotomy of left internal rectus muscle and transplantation	None on cover test	25°	Adduction to 35°

operation, and in this case, to be discussed later in the paper, the resulting inconvenience was slight. This is to be emphasized, as it is undoubtedly fear of this complication which has prevented many surgeons from undertaking the operation.

Case 6 illustrates the serious disadvantage of postponing operation until marked contracture of the opponent has developed, a point emphasized by O'Connor. The patient had suffered paralysis of the external rectus muscles following a blow on the head seven years before. Contracture was extreme, especially of the left internal rectus muscle, so that the convergence of this eye, which was 50 degrees, could be reduced to only 40 degrees by an extreme effort of abduction. Deviation of the left eye was 30 degrees and could be reduced to only 20 degrees when the patient looked to the right. The total angle of squint was 80 degrees. Bilateral transplantation with free tenotomy of both internal

Fig. 2 (case 6).—*A*, appearance of the patient before operation. *B*, appearance after operation, with eyes front and left convergence of 15 degrees. *C*, appearance after operation, with eyes to the right and the right eye abducted 15 degrees.

muscles was performed,[12] but it was impossible to correct the convergence entirely, even by a second operation, at which the left internal rectus muscle was advanced and the eye was held in the corrected position by tension sutures. The cosmetic improvement was great, however, and abduction of the right eye to 15 degrees is possible, while the left eye can be brought only to the midline (fig. 2). The tendon slips were found at the second operation to be in proper position. A left hypotropia of 10 degrees is present.

In case 3 transplantation and recession of the internal rectus muscle produced an insufficient effect. Later tenotomy of the internal rectus muscle and advancement of the paretic external muscle produced an overeffect, with divergence of 10 degrees. In case 5 the result of transplantation and tenotomy of the internal rectus muscle was a marked

12. One operation was performed by Dr. William Mann.

overeffect (15 degrees divergence). This showed no signs of improvement during three months, so the internal rectus muscle was advanced 3 mm. in front of its previous insertion. The result of this procedure was quite satisfactory, with no deviation on cover test and 20 degrees of abduction. In 1 other case not listed in the table, an overeffect

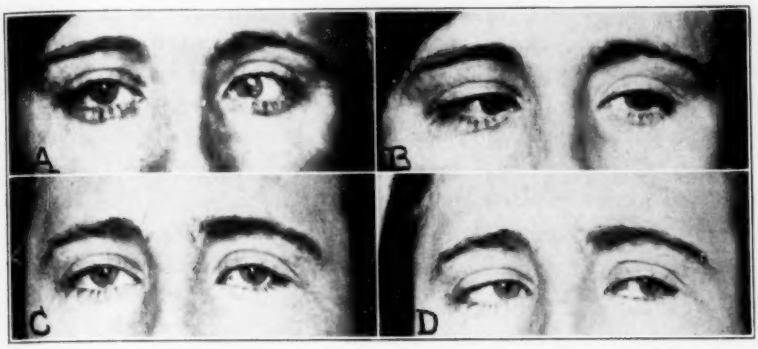

Fig. 3 (case 7).—*A*, appearance of the patient before operation, with eyes front. *B*, appearance before operation, with eyes left. *C*, appearance after operation, with eyes front. *D*, appearance after operation, with eyes left.

Fig. 4 (case 2).—*A*, appearance of the patient before operation, with eyes front. *B*, appearance before operation, with eyes left and marked secondary deviation of the right eye. *C*, appearance after operation, with eyes front. *D*, appearance after operation, with eyes slightly left. (The left eye can be abducted somewhat farther alone.)

was produced by transplantation combined with tenotomy, while in case 10, although the cosmetic result is satisfactory, convergence is somewhat limited. In case 1 a marked overeffect followed the operation, but this became less; after two months the result was satisfactory.

Operation in case 7 was done elsewhere without effect. Transplantation and recession gave a good cosmetic and functional result (fig. 3). Case 2 presented interesting features. The primary deviation was only 15 degrees and was compensated for by keeping the head turned to the left. When the patient attempted to look to the left, as soon as the midline was reached the paralyzed left eye at once assumed fixation, while the right eye converged markedly due to secondary deviation. It was this phenomenon and the head turning necessary to prevent it which brought the patient in for correction. A previous operation elsewhere had brought about improvement for about one year, after which the condition became as before. At operation the internal rectus muscle was found to be 12 mm. back of the limbus and was not disturbed. The external rectus muscle, which had presumably been advanced, was found to be 11 mm. back of the limbus. It was not disturbed, and the transplanted slips were anchored in a scleral groove prepared 5.5 mm. from the limbus. The patient now has no primary deviation and holds the head straight. She can look to the

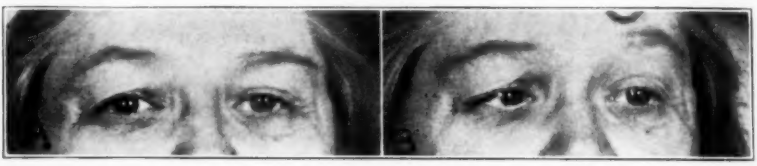

Fig. 5 (case 10).—*A*, appearance of the patient after operation with eyes front. *B*, appearance after operation, with eyes left.

right at least 7½ degrees without secondary deviation of the right eye, can abduct the left eye alone 15 degrees and is comfortable (fig. 4).

The result in case 10 (fig. 5) somewhat resembles that in case 2. The patient can abduct the left eye to 25 degrees without diplopia. Beyond this point, the left eye fixes, while the right eye converges markedly due to secondary deviation. Adduction is somewhat limited, as was true in several cases, including case 11.

That transplantation alone is capable of producing considerable effect is shown by several of O'Connor's cases and by case 2. It might seem safer to perform transplantation first and follow it by recession later if the effect was not sufficient. Experience with other types of squint has shown, however, how much greater is the effect of a shortening operation when combined at the same time with weakening of the opponent, and it would seem that in cases in which there is primary deviation of over 15 degrees recession of the internal rectus muscle at the time of transplantation should be performed if enough effect is to be obtained.

In a case not listed in the table transplantation alone was performed without any interference with the internal rectus muscle. The effect was insufficient to correct the primary deviation.

It will be noted that cases 3, 5 and 9 were cases of congenital deficiency of the external rectus muscle with Duane's retraction syndrome. The retraction was not especially marked in these cases, but it is interesting that no abnormal bands or attachments were found when the internal rectus muscle was freely exposed and freed. The conditions in cases 7, 11 and 12 were of congenital origin but without the retraction syndrome.

During this period I have seen several cases of congenital deficiency of the external rectus muscle in which the primary deviation was so slight as to be inconspicuous, and in these cases transplantation was not considered. In 1 of these cases a marked retraction syndrome was present with marked up-shoot of the left eye on attempted adduction. In this case tenotomy of the inferior oblique muscle has greatly reduced this up-shoot, and the patient, now 5 years old, is being observed to see if contracture of the internal rectus muscle will develop.

CONCLUSIONS

Experience with these cases has led me to the following conclusions:

1. Transplantation of living tendon slips from the superior and inferior rectus muscles offers the best chance of a cosmetic and functional result in paralysis of the external rectus muscle. The outer halves of the muscles were chosen in these cases, and the results, on the whole, were satisfactory.

2. Operation should, as a rule, be accompanied by recession of the internal rectus muscle. In cases with primary deviation of over 15 degrees a recession of 5 mm. is usually safe.

3. Overeffects are rare and can usually be overcome by replacing the internal rectus muscle.

4. Tenotomy of the internal rectus muscle should be reserved for cases in which there is marked contracture of that muscle.

5. No vertical imbalances or deviations occurred as a result of the operation.

6. The operation should be performed before secondary contracture has occurred. In cases of acquired paralysis if no improvement has occurred after three to six months of observation, and especially if the paralysis is increasing, operation is indicated. In cases of congenital paralysis it may be safely performed at the age of 3 to 5 years.

7. In answer to Bielschowsky's criticism, it may be stated that all the patients except those in cases 3 and 6 have a useful field of binocular fixation while holding the head straight and do not hold the head in an abnormal position.

MASSACHUSETTS VISION TEST

AN IMPROVED METHOD OF TESTING EYES OF SCHOOL CHILDREN

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The need for a complete ocular examination for all school children by a qualified ophthalmologist is recognized. Until such a time as practicable means can be found for this to be realized, it is necessary to rely on some procedure in the school which will screen out those who need particular attention.

It is of utmost importance that the ophthalmologist should take the initiative in the designing of screening device procedures and should not allow this responsibility to fall outside the medical profession. The scope of such work is so broad and involves so many issues other than construction of a kit and designing criteria that the closest cooperation with the educators and psychologists is necessary for the most effective results.

In Massachusetts the staff of the Research Learning Project had an unusually favorable contact with a number of rural and urban districts in which an extensive investigation into certain factors affecting learning had been under way for a year. The collaboration of medical, educational, psychologic and administrative specialists made possible the handling of large groups with great efficiency, saving of time and excellent cooperation.

Throughout the progress of the study, a particular effort was made to combine psychologic and educational theory with ophthalmologic knowledge and practice.

DEVELOPMENT OF THE MASSACHUSETTS VISION TEST

The staff of the Research Learning Project turned its attention to the development of a screening test during the school year 1938-1939. The following premises became our objectives:

Read before the Section on Ophthalmology at the Ninety-First Annual Session of the American Medical Association, New York, June 13, 1940.

This study was made by the staff of the Research Learning Project, Division of Child Hygiene, M. Luise Diez, M.D., director. In addition to Dr. Sloane, the staff consists of the following: Lura Oak, head of the Research Learning Project; Miss Miriam Forster, research assistant; Philip W. Johnston, research consultant, and John W. M. Rothney, statistician. The study was undertaken as a subsidiary investigation incidental to the main purpose of the project, which is to study the causes of early failure in school.

1. Effective application by nonprofessional persons.

2. A method of scoring that would not appear to indicate the degree of defect to the lay person administering the test.

3. Simplicity of materials and of administration.

4. Brevity.

5. Duplication, as far as possible, of conditions and methods commonly used in the office of an ophthalmologist.

6. Economy in the cost of materials consistent with efficiency.

7. The inclusion of tests to detect latent errors in binocularity.

8. The inclusion of tests to detect latent errors in refraction as well as reduced vision.

9. The standardization of artificial illumination of the test chart.

10. A policy of referring for examination the children with so-called "borderline" or questionable cases so that the ophthalmologist may be the one to pass judgment as to whether or not treatment is required.

The original screening test employed, in addition to the present components, the following features, which have now been omitted:

1. A measure of positive, relative, accommodation. A pair of —3.00 D. spheres mounted in zylonite frames were utilized in the following manner: The 20/20 line was fixed and read through these glasses. We found, however, that many children who had an amplitude of over 15 D. of accommodation could not overcome —3.00 D. spheres at distance. There seemed to be a knack in ignoring the sense of distance in order to overcome these lenses which many could not learn on first trial and yet could later do with great ease.

2. A measure of prism convergence. A pair of prisms of 6 prism diopters was mounted base out in zylonite frames. A distant 20/200 letter was fixed and seen singly if the influence of the prisms was successfully overcome. In 350 cases this test screened out only 1 child, so that it was felt that it could be omitted, thus simplifying the battery.

3. Stereoscopic methods of determining single binocular vision and phorias. Single binocular vision was tested by using the split E card. The horizontal phoria was evaluated by utilizing a card which contained a properly calibrated rectangular enclosure before the right eye and a small ball before the left eye.

The vertical phoria was ascertained by making the diameter of the ball of such a size that if the hyperphoria exceeded normal, the ball would be displaced above or below a horizontal line. A different card was arranged for testing near and distant vision. Infinity and 16 inches (40 cm.) were arrived at by placing the card at the optical equivalents on the stereoscope shaft. We soon decided that the stereoscopic method was a misleading one when employed by the lay person.

The following observations were noted and probably explain many of its shortcomings:

(*a*) The stereoscope while optically set for infinity was too frequently influenced by a psychologic sense of nearness.

(*b*) A poorly made stereoscope introduces errors, and a good stereoscope is relatively expensive.

(*c*) In the hands of lay persons there were too many observations made by the child which were confusing to the interpreter.

(*d*) In keeping with our aim of duplicating methods used by the ophthalmologist in his office, we found that the Maddox rod test was more commonly used than stereoscopic methods. Statistically, we compared the results with the Maddox rod method and those with the stereoscopic method in 200 cases. In all instances the response was called questionable when it was not clearcut and caused some question of interpretation. The results of this comparison follow:

No. examined by routine phoria test (staff ophthalmologist)	200
No. passed	181
No. failed	19
Results for the 181 passed by ophthalmologist:	
With the Maddox rod (by technician):	
No. passed	179
No. failed	2
No. with questionable response	0
With the stereoscope:	
No. passed	110
No. failed	7
No. with questionable response	64
Results for the 19 failed by ophthalmologist:	
With the Maddox rod (by technician):	
No. passed	4
No. failed	15
No. with questionable response	0
With the stereoscope:	
No. passed	2
No. failed	12
No. with questionable response	5

It was found that the responses to the Maddox rod test were decisive and left a minimum of confusion in the mind of the lay person testing.

THE MASSACHUSETTS VISION TEST

The Massachusetts vision test is made up of three tests, to be given in sequence.

1. *The Visual Acuity Test.*—The findings for the visual acuity test in terms of the Snellen fraction are expressed for (*a*) the right eye, (*b*) the left eye and (*c*) both eyes. Failure with this test is an indication to omit the remaining tests.

2. *The + 1.50 Sphere Test.*—For this test a pair of + 1.50 spheres are mounted in zylonite spectacles. The child is instructed to close his eyes and then to open them and read the 20/30 line. If this is accomplished, the 20/20 line is tried. This test is applied to (*a*) the right eye, (*b*) the left eye and (*c*) both eyes. Failure with one of these tests is an indication to omit the remaining tests.

3. *Heterophoria Test.*—For this test two pairs of zylonite spectacles are supplied, each containing a red multiple Maddox rod for the right eye and no lens for the left eye. One Maddox rod is mounted horizontally and the other vertically. The test is made at 20 feet (6 meters), and the object of fixation is a small 5 watt nonfrosted bulb which projects through the window of the picture of a house. The lateral dimensions of the house are calibrated to be equivalent to 6 prisms of esophoria and 4 prisms of exophoria for a distance of 20 feet. The vertical dimensions of the window are calibrated to be equivalent to 1.25 prisms of right and left hyperphoria.

In a similar way the near phoria test is made by utilizing a block of wood in which is concealed a pencil type of flashlight which furnishes a source of illumination through a pinhole aperture. The width of the block is calibrated for 6 prisms of esophoria and 8 prisms of exophoria. To the block is attached a 16 inch cord for measuring the proper distance. The face of the block has a small picture for fixation so that accommodation will be in use. The presence of the vertical streak on the block constitutes a successful performance. Thus, this device tests (*a*) vertical phoria at 20 feet, (*b*) horizontal phoria at 20 feet and (*c*) horizontal phoria at 16 inches. Failure with one part of the test is an indication to omit the remaining tests.

ADMINISTRATION OF THE TEST

Test 1 is administered according to the following instructions:

(*a*) Cover the left eye and ask the child to read the first line in the first box. (If he is successful, record the smallest letters read.)

(*b*) Cover the right eye and have the child read the first line in the second box.

(*c*) Tell the child to open both eyes and read the first line in the third box.

NOTE.—If the three parts score 20/20, proceed with test 2. If visual acuity is less than 20/20 with any one of the three parts of this test, the remaining tests are omitted.

Test 2 is administered as follows:

(*a*) Have the child close both eyes after the spectacles are placed. Tell him to read the second line (20/30) in the first box. (If he is successful, have him read the second line in the second box.)

(*b*) Have the child repeat (*a*) with the right eye closed.

(*c*) Have the child repeat (*a*) with both eyes open.

Failure with any part of the test is an indication to omit the remaining tests.

Test 3 is administered as follows:

(*a*) Place the Maddox rod spectacles with "vertical ribs" on the child's face. Briefly, the following instructions are given:

"Cover the left eye."

"Do you see a red streak?"

"Does it go up and down or cross-ways?"

"Uncover the left eye."

"Do you see the streak and the house?"

"Does the streak pass through the window or elsewhere?"

(*b*) Place the Maddox rod spectacles with the "horizontal ribs" on the child's face and repeat as in (*a*), except for the last question: "Does the streak pass through the house or elsewhere?"

(*c*) With the Maddox rod with the "horizontal ribs" on the child's face, have him fix the small picture on the near phoria testing block and ask: "Does the streak pass through the block or elsewhere?"

NOTE.—Whenever the child's response is not clear as to the location of the streak, the spectacles are removed and he is asked to point on the block or walk to the picture of the house to indicate the position of the streak.

SCORING FOR TEST AND TIME REQUIRED

On failure with any one test, the remaining tests are omitted. This will serve (*a*) to shorten the time needed for the test and will (*b*) avoid confusing problems with the heterophoria test. (Example: Amblyopia in one eye will result in the streak and house not being seen at the same time.)

With test 1, if the visual acuity is less than 20/20 — 2 further examination is indicated (F.).

With test 2, ability to read 20/20 is classified as failure (F.) and inability to read 20/30 as passed (P.). Ability to read 20/30 but not 20/20 is considered a partial failure (P. F.) monocularly and of no significance binocularly. Two partial failures (P. F.) are equivalent to failure (F.).

With test 3, the following classifications are employed:

(*a*) If the child sees the streak through the window he passes (P.); if he does not see it through the window he fails (F.).

(*b*) If the child sees the streak pass through the house he passes (P.); if he does not see it pass through the house he fails (F.).

(*c*) If the child sees the streak pass between the lateral limits of the block he passes (P.); if it passes elsewhere he fails (F.).

From the third grade level up it requires less than three minutes to give the test. It takes slightly longer for slower children and considerably shorter for brighter children.

FEATURES OF THE TEST

Illumination.—The test chart is illuminated by two 25 watt bulbs, giving an intensity of about 16 foot candles. Haphazard window illumination is no longer a factor.

The Test Card.—Three rectangular enclosures each contain a different 20 and 30 foot line. In our most recent charts the same letters are used in a shuffled order. There are 8 letters to each 20 foot line. Three different 20 foot lines tend to minimize memorization. A fourth rectangular enclosure contains 4 letters of graduated size (20/100, 20/70, 20/50 and 20/40) whereby visual acuity may be recorded.

Duplication of Office Procedure.—A 20 foot distance is required. The Maddox rod is used to test phorias. The examiner faces the child during the test.

Training of the Teachers.—A short course in basic knowledge concerning refraction and other conditions of the eye is given to the teacher. In our experience we find that when properly administered the Snellen test is the single most effective procedure for detecting children who require the attention of an ophthalmologist. However, there are great discrepancies when the test is given by the teachers and by the ophthalmologist. In tabulating the Snellen visual acuity records of the teachers with those of the staff ophthalmologist in 387 cases, there was agreement in 270 and disagreement in 119 cases (30 per cent). Lack of basic training and care in giving the tests as one cause for this discrepancy is suggested by a comparison of a trained technician's Snellen records as compared with the staff ophthalmologist's. In 288 cases there was agreement in 90 per cent and disagreement in 10 per cent (30 cases).

Degree of Error.—It is stressed that the decisiveness or ease with which one fails to pass a test is no indication of the degree of error. We have found that in certain instances lay persons testing sight have assumed to evaluate defects. (Example: 20/200 = 10 per cent vision or 90 per cent blind.)

Stopping the Test.—When failure occurs, the remaining tests are omitted.

Failing Visual Acuity.—It is our impression that with good illumination vision of 20/15 is a common finding. For this reason we have accepted 20/20 — 2 as the limit for passing. We appreciate that a number of children will be sent unnecessarily to the specialist for further examination. Nevertheless, a considerable number needing attention but ordinarily missed by the usual Snellen test will be detected.

Retesting.—It is our suggestion that all children who fail be retested by a second person, who shall make the final decision before the child is referred to an ophthalmologist.

Individual Examination.—Only one child is in the room at a time. In this way memorization is further avoided.

PROBLEMS PRESENTED BY EXAMINATION

Amount of Heterophoria.—It is appreciated that the amount of imbalance set as a criterion for referring the child to an ophthalmologist is debatable. There will be the objection that it is too wide and too narrow. However, since the status of degrees of heterophoria varies with different examiners and localities, we hope that a happy medium will have been found. The present arrangement permits of modification.

Amount of Hypermetropia.—The plus sphere test is open to controversy. The amount of plus sphere (1.50) may be criticized as being too much or too little. We must agree that there are a number of persons with hypermetropia of + 1.50 who will not relax their accommodation and consequently will be unable to see through the lenses.

Mechanical Problems.—The equipment must be perfected so that it will not be affected by wear and tear too soon. The near phoria test block can stand improvements in construction. We hope these problems will be solved by the manufacturers who have experience and facilities for dealing with such programs and are better equipped to carry this burden of the program.

THE EXPERIMENTAL PROGRAM

Program In Massachusetts for 1938 to 1939.—Three hundred and thirteen school children between the ages of 7 and 15 were examined with and without cycloplegia by the staff ophthalmologist. The tests were carried out in the public schools of Newton, Walpole and Barnstable. The group represented a random selection of children from rural and urban sections. A decision was reached by the ophthalmologist as to whether the child did or did not require the services of an ophthalmologist. In instances in which there was doubt as to the need for the child's being referred, the term "borderline" was used. It was found that there were sufficiently complete records in 248 cases to warrant

comparison. Sixty-five cases were not included in the report because the records were either incomplete or marked incorrectly. The Massachusetts vision test was given independently by another member of our staff. The comparison of the records of the ophthalmologist and the technician were made by a third person. In brief, the results were:

Totol no. of children examined 248
No. passed by ophthalmologist ..... 161, or 65%
No. failed by ophthalmologist ..... 87, or 35%

Results with Massachusetts vision test:

For the 161 passed by the ophthalmologist:

No. passed ..... 138, or 56%
No. failed ..... 23, or 9%

For the 87 failed by the ophthalmologist:

No. passed ..... 6, or 2%
No. failed ..... 81, or 33%

Results with other tests for the 23 failed unnecessarily by the Massachusetts vision test:

No. failed with Snellen test 16
No. failed with Maddox rod test 2
No. failed with + 1.50 sphere test 5

It must be recognized that these tests were given by one technician and one ophthalmologist.

Program In Massachusetts for 1939 to 1940.—The second part of our experimental program provided for the following:

1. The tests were to be given by teachers and nurses who ordinarily in the past have given the Snellen test. The following centers were chosen: Barnstable, Weston, Walpole, Amherst, East Longmeadow and Newton. All the teachers and nurses participating in this program attended three lectures where the tests were demonstrated, and an elementary consideration of the following subjects was discussed: (*a*) anatomy and physiology of the eye, (*b*) hypermetropia, (*c*) myopia, (*d*) astigmatism, (*e*) strabismus, (*f*) heterophoria and (*g*) symptoms and signs suggesting ocular difficulty.

2. Each child who failed was to be retested by a second person, usually the school nurse, before the child was referred to the family specialist. The results were:

Total no. of children tested 3,430
Percentage of failure with the test when given by teachers (according to district in which test was given) ..... between 39 and 40%
Percentage of failure for the entire group 43%

Percentage of failure with different parts of the test for the entire group:

Test 1	32%
Test 2	7%
Test 3	4%

NOTE.—If the same group of 3,430 children had been scored with 20/30 passing for test 1, the percentage of failures would have been 29 per cent.

Out-of-State Program.—Dr. Luedde, Dr. Vail and Dr. Gifford offered to have the Massachusetts vision test given in their respective cities to a limited number of children. A technician was instructed in their use, and each child was independently examined in the routine manner by an ophthalmologist. At the time this paper was written, the results of Dr. Luedde's group only were available.

Total no. of children tested (Drs. Luedde and Drews)	90
No. failed by ophthalmologist because of a refractive error	19
No. of these failed with Massachusetts vision test	15, or 79%
No. failed by ophthalmologist because of heterophoria	2
No. of these failed with Massachusetts vision test	1, or 50%
No. passed by ophthalmologist	69
No. of these passed with Massachusetts vision test	46, or 67%
No. of these failed with Massachusetts vision test	23, or 33%

Thus, 23 per cent were failed by the ophthalmologist and 43 per cent failed with the Massachusetts vision test. (In this instance the figures are based on only one administration of the test.)

Special Program at Weston.—A special study was made at Weston in which 278 tests were rescored on the following basis:

1. Section (*c*) was omitted from tests 1 and 2.

2. Visual acuity of 20/30 in the right or left eye was scored as P. F. in test 1.

3. P. F.'s were combined between tests 1 and 2 so that an accumulation of two P. F.'s in any way whatsoever resulted in failure.

4. The figures were corrected for those children who would have failed if the test had been continued.

5. The remaining tests were scored in the original way.

The results at Weston were:

No. of children failed with test 1	21%
No. of children failed with test 2	5%
No. of children failed with test 3	2%
Percentage of failure for the entire test, when scored this way	28%

PROBLEMS TO BE SOLVED

Criteria for Passing.—1. Visual Acuity: In most states visual acuity of 20/30 is considered passing. In the experience of most ophthalmologists it is noted that 20/15 is not an uncommon normal finding.

Similarly, many fairly high refractive errors are found associated with naked vision of 20/30. Since it is our premise to err in the direction of referring more children than necessary to the ophthalmologist, rather than to miss gross defects, we have set 20/20 — 2 as a passing requirement. We realize that many will feel that this is too high a requirement. Modification can be made later if necessary. Vision of 20/25 may prove to be a more "happy medium."

2. Heterophoria: This is generally accepted as a condition requiring investigation. Unfortunately there is no real consensus as to how much an error must be present before it is deserving of attention. Opinions favor both extremes. Since such is the situation, we must expect a certain amount of dissension regarding the limits we have set for passing. In its present state the Massachusetts vision test is calibrated so that 6 prisms of esophoria and 4 prisms of exophoria for distance, 8 prisms of exophoria and 6 prisms of esophoria for near vision and 1.25 prisms of hyperphoria are the limits for passing. We appreciate the shortcomings of the Maddox rod test and the advantages of certain other tests, including the great potentiality of utilization of polaroids. At this time, however, the Maddox rod has been demonstrated to be a simple method in the hands of the lay person.

3. Latent Hypermetropia: The plus sphere test is hardly the perfect test for uncovering latent hypermetropia. In the hands of many persons who have given the test, it is a substitute that is not without value. The amount that we have specified as failing (1.50) is subject to controversy. In our hands it seems to be the "happy medium." It is likely that a stronger lens may be needed up to age 8 and 9 and possibly a weaker lens at the high school age.

4. Role of the Ophthalmologist: Under this heading are four great issues:

(*a*) It is certainly advisable for every school child to be examined thoroughly by a qualified ophthalmologist. At present there are not enough ophthalmologists to handle the school population adequately.

(*b*) It is unfortunate that in many instances tests for binocular functions are not included in the regular examination. No doubt the ever-increasing demands for higher qualifications prior to practice of a specialty will correct this situation.

(*c*) The ophthalmologist must assume a certain amount of responsibility in all programs involving ocular tests in the schools. This cannot be left entirely to nonmedical persons. The cooperation of educators and medical men is of invaluable importance, since each has a certain part to play.

(*d*) Unfortunately, there can be no control as to what kind of "eye doctor" the children should visit. The most conscientiously planned

public health program becomes detrimental if it succeeds in bringing the child to a charlatan.

Miscellaneous Problems.—Other problems to be solved may be visualized in a topical outline such as the following:

- Training of persons to give the tests
  - By lecture or pamphlet
  - In training schools
  - (Results are better when trained people give the test.)
- Administrative problems
  - Burdens of the teachers
  - Lack of substitute teachers
  - Physical shortcomings
  - (Example: No room for test.)
- Who shall give the test?
  - Teachers
  - Nurses
  - Specially assigned persons
  - Problems involved
- Equipment
  - Costs
  - Less unwieldly
  - Foolproof
  - Transportable
  - Mechanical improvements

CONCLUSIONS AND SUMMARY

There is definite need for a screening test.

Present methods of screening are inadequate.

It should be the premise of the ophthalmologist to design and take an active interest in screening tests rather than to allow the responsibility to fall outside the medical profession.

The cooperation of the educators is necessary toward effective development of screening tests.

The Massachusetts vision test has as its objectives: (1) effective application by nonprofessional persons, (2) a method of scoring that would not indicate the degree of defect to the lay person administering the test, (3) simplicity, (4) brevity, so that great numbers may be tested, (5) duplication, as far as possible, of conditions and methods as commonly used in the office of an ophthalmologist, (6) economy, (7) detection of latent errors in binocularity, (8) detection of latent errors in refraction as well as reduced vision, (9) standardized artificial illumination of the test charts and (10) reference for examination of children with questionable cases so that an ophthalmologist may pass judgment as to whether or not treatment is required.

The Massachusetts vision test includes a test for (*a*) visual acuity, (*b*) heterophoria at distance and near vision and (*c*) latent hypermetropia.

The Massachusetts vision test provides for standardized illumination, a special test card and certain other features.

A wide experimental program has formed a background for the Massachusetts vision test.

The results indicate that the Massachusetts vision test tends to screen out more effectively than the single Snellen test.

As in any subjective test like the Snellen test, the results are most reliable when the tests are given by persons with special aptitude and skill in their administration.

In a similar way, in the hands of teachers who are less proficient in giving the test, the results are not as effective.

The Massachusetts vision tests utilize limits for passing in visual acuity, hypermetropia and heterophoria which we believe are a "happy medium." They may be modified if desirable.

The administrative problems may be multiple and complex in the initial stages of introducing the tests into a given school.

Education relating to the eye and its testing should be included in the curriculums of teachers' training schools.

Certain qualified persons should be selected eventually to give the tests.

Visual acuity of 20/20 as a requirement for passing is probably too high.

A different method of scoring the same tests may be desirable.

There are many problems to be solved.

Further research is needed in the field of testing vision of school children.

The present study must not be considered complete or final.

Dr. Walter B. Lancaster gave invaluable advice and assistance.

ABSTRACT OF DISCUSSION

DR. S. JUDD BEACH, Portland, Me.: The distinct advance portrayed in Dr. Sloane's report is the experimental evaluation of the means to be used for selecting children to be sent to specialists for examination of the eyes. In the past, lack of proper interpretation has detracted from methods otherwise possessing merit. As a result, many children were either referred needlessly or overlooked.

In the last dozen years there have been three different ways of handling this problem. The first has been to expedite the use of the Snellen card in order to minimize the interference with school work.

It was at the Minneapolis session of this Section that Harrington's lantern was shown, which examined, I think, 15 children at one time;

the next year Roden's plan was presented, and more recently Morrissey's rapid visual screening test has been developed.

As Dr. Sloane said, because of the numbers of children who elude the Snellen test, since it tests only central visual acuity, attempts have been made to include examination of other functions. In the procedure that was outlined by the National Education Association and the American Medical Association group this is done indirectly by noting indications of ocular dysfunction, such as redness, and headache, while in that of the Student Health Association and the Betts test and this method which is now under consideration special tests of other functions are used.

Of course the difficulties of committing to the laity the investigation and detection of symptoms and of entrusting to them the investigation of intricate functions, as by the use of fusion tests, are perfectly obvious.

Dr. Sloane's outstanding contribution has been his evaluation of these tests by actual experience before permitting their general use. He has resisted several invitations, to my knowledge, to present them prematurely, among them one before the preceding meeting of this Section.

It is unnecessary to point out the importance of having work so intimately connected with the general health performed by medical experts, and encouraging as his results are, they are less significant than the judicial approach by which he has enabled the medical profession to assume its responsibility for devising and evaluating this procedure.

Dr. Maynard C. Wheeler, New York: We all agree with Dr. Sloane that it is the responsibility of the ophthalmologist to establish this work of screening out the children who require ocular examination, but I wonder if many have taken an active part in the work. I should like to confine my remarks to the measurement of muscle anomalies. The plan of omitting subsequent tests when the child has failed to pass the visual test is an admirable one in the interest of saving time and avoiding a great deal of confusion with the Maddox rod test.

Dr. Sloane has hinted at the shortcomings of the Maddox rod in measuring phorias. I should like to emphasize that. Many ophthalmologists still rely largely on the Maddox rod for the diagnosis and measurement of all phorias. Fortunately, increasing numbers of ophthalmologists are appreciating the inaccuracy of this test and are taking up other methods of measuring the muscle anomaly and particularly are utilizing the cover test, which many agree is a great deal more accurate and does not add any complications to the routine examination.

The recent examinations of the American Board of Ophthalmology have demonstrated that the Maddox rod test is not foolproof, even in the hands of ophthalmologists. I will confess that it is hard to find a better test than the Maddox rod test for the lay examiner, but I would suggest that rapid screening can be combined with the Maddox rod test, the two eyes being exposed only for brief moments, without complicating the test hopelessly and certainly increasing its accuracy considerably.

I do not think Dr. Sloane read that part of his paper in which he more or less anticipated objections to his criteria for the normal muscle findings. I am not going to disappoint him in this regard, because I do object to the standards that he has selected. I believe that they are too high, particularly for the esophorias. Authorities vary in their figures for the normal findings, but to mention only two of them, Duane and

Dunnington will not allow any esophoria for near vision, yet Dr. Sloane placed 6 diopters as his normal limit. Perhaps that difference has arisen from his use of the Maddox rod, which tends to give high measurements for esophoria. However, these criticisms in no way invalidate the splendid results that he has obtained.

DR. S. WEIR NEWMAYER, Philadelphia: One cannot overestimate the importance of these tests, because they have a direct influence on acquiring an education.

One should keep in mind the chief objectives for testing eyes of school children: (1) to assure educators that each child has ample visual acuity so that he can progress in a normal school life; (2) to prevent progress of pathologic conditions by their early recognition and (3) to help the visually handicapped by early diagnoses of their errors, so that they can be placed in special classes.

Many important problems arise in some towns and cities and make a variable program necessary. Pennsylvania has a school law that makes a complete physical examination of each child at least once a year mandatory. In Philadelphia, with a school enrolment of about 400,000 and about 3,500 children assigned to each medical examiner, the possible time to be alloted to eyes alone is limited. Visual acuity taken on test charts is available and limited in diagnostic value. The use of instruments of precision requires much time and services of specialists.

Modern education has abandoned teaching letter recognition and the alphabet; so few children in lower grades can be tested by letter charts. I have found that charts composed of numerals prove more practical.

Screening of children for further study by oculists is often accomplished by observant teachers. Such pupils are chosen according to their ability to learn to read. Various cities report from 25 to 50 per cent of their school population as poor readers or nonreaders. About 5,000 children are referred to me each year, to assign a cause for failure in reading. The failure of about 50 per cent is due to ocular defects and of 50 per cent to faulty technic in teaching.

I agree with Dr. Sloane's comments about failure in the use of stereoscopic instruments for diagnosis. The testing of muscles in school tests is not practical and diagnostic by any method. It permits too many fallacies. I agree that the visual acuity is not always a safe diagnostic sign in a child. Frequently children with recorded normal vision are poor readers, and ophthalmoscopic examination by an ophthalmologist finds the child has marked or important defects. A child may readily overcome 3 or 4 diopters of hyperopia or 2 diopters of myopia and often some astigmatism by use of its accommodation.

I believe the practical solution of the ocular problems in a public school system is the use of the experienced ophthalmologist who has trained himself in the use of the ophthalmoscope so that he can quickly recognize defects of refraction as well as major pathologic processes. He can decide whether the child is one to be further studied by the private ophthalmologist or at a clinic in order to be assured of appropriate treatment.

DR. EDWARD JACKSON, Denver: The testing of the vision of school children has great medical significance, but it is also a fundamental requirement of justice to the school children. It is known that great variations in ability to do school work depend on variations in visual

distinctness or visual effort. In the testing of school children one has an opportunity of educating the whole public as to the requirements of good vision and as to what is good vision and what is easy vision.

There are one or two points that have not been alluded to in the report but have stood as obstacles to doing this work. One is that many of the children are not acquainted with the letters of the alphabet at the time when the testing would be most valuable to them and to the community. Any child's vision can be tested with the incomplete square of Snellen.

Snellen, in the second edition of his test type, introduced the incomplete square—the square with one side left out. This offers four tests to any child, and if the child cannot tell right or left he can point with his finger.

Another point that is of great practical importance is that there is no good test for excessive hyperopia—farsightedness. If the error does not interfere with the near use of the eye, it is not a serious matter. However, any interference which makes focusing of the eye excessively difficult creates a disability for school work.

There is an objective test that has been used for this purpose. I have often been conscious of its value in the last two or three years, and it ought to be considered in every system of testing the eyes of school children. For this test one takes either fine letters, if the child knows the fine letters, or finely printed figures. More young children can recognize the figures that they see on the street cars or that they see in any computation of change. If they understand numerals, nine numeral figures furnish a sufficient list for the testing of near vision.

Take any figure or any picture that interests the child and hold it before his eyes; bring it slowly toward the eyes, and ask the child about the details. Presently he will begin to rear back his head. It is easy to measure the distance and it is easy to keep the child's interest so that the test can be repeated two, three or half a dozen times. In this way one can be sure at what point the child's head begins to retreat.

I have seen 2 sisters who reached the seventh and eighth grades in school who had 15 diopters of hyperopia in each eye. If a child has an excessive hyperopia, it may be only a diopter or so; but if he feels the extra effort required of the eyes, the drawing back of the head can easily be recognized. This test can be applied even by young teachers to the youngest children in the school. It can be applied intelligently by any physician, and it brings out the great majority of refractive errors in young children—the hyperopias and the hyperopic astigmatisms. I know of no other test that will reveal it.

DR. ALBERT E. SLOANE, Boston: I should like to emphasize that this is a preliminary report and must not be considered final.

In regard to Dr. Wheeler's point relating to the presence of esophoria on use of the eyes for near vision, the test is so arranged that when some decision can be reached by the ophthalmologists of the country one can modify the amount of failing phoria. For the present, it must be remembered that there must be practicability in the numbers of children referred to an ophthalmologist. If 90 per cent of the school population are referred, the test will not meet with approval.

Dr. Newmayer made some suggestions about nonreaders. Not knowing anything about the reading problem except what I hear, I can-

not discuss that; but I feel that every child who is handicapped in his reading is entitled to a complete examination by the ophthalmologist and should be referred for examination whether he passes or does not pass the screening test.

Dr. Jackson mentioned the need for a chart using illiterate symbols. We have a chart which utilizes the illiterate symbol E.

I wish I were as optimistic as he is about showing teachers how to make an objective test, but in my carefully considered opinion the average teacher cannot be taught to give an effective cover test. I also think that it would be very difficult for the teacher to give a test for the near point of accommodation and to observe accurately movements of the eyes.

I hope that some means will be acquired of having specially trained technicians give the recheck test so that some finer tests can be added to discover defects which might otherwise be missed, but for the present the more subjective the tests are, the better.

PRECHIASMAL SYNDROME PRODUCED BY CHRONIC LOCAL ARACHNOIDITIS

REPORT OF THREE CASES

WALTER I. LILLIE, M.D.
PHILADELPHIA

Prechiasmal and chiasmal inflammatory lesions which produce a slowly progressive syndrome suggestive of tumor can be broadly placed in two groups, namely: (1) basal gummatous meningitis and (2) chronic local arachnoiditis. The former can be diagnosed readily by the serologic and cytologic changes in the cerebrospinal fluid and needs no further consideration here. The latter group usually presents a clinical syndrome so like tumor similarly situated that it is almost impossible to predicate the underlying lesion. Surgical exploration reveals the optic nerve or the chiasm to be covered with definite inflammatory exudates and bound down by a single adhesion or a mass of adhesions. Tumefaction of the inflammatory exudate, which consists of encysted fluid, may also be present. The type of pathologic process found will depend on whether the clinical ophthalmologic findings suggest a space-taking lesion, similar to a basofrontal tumor, or a chiasmal lesion, such as an infrasellar or a suprasellar tumor.

Because it is extremely difficult to predicate the underlying pathologic lesion from the ophthalmologic findings alone, other clinical measures are usually sought. The roentgenographic findings in chronic basal arachnoiditis always reveal a normal sella turcica, without evidence of bone erosion or exostoses and no calcification in the immediate neighborhood. This in itself is not sufficient evidence to make a diagnosis of chronic basal arachnoiditis, as many space-taking lesions can and do give a normal roentgenographic picture. Although the defects of the visual field are characteristic of a lesion affecting the visual pathways in this vicinity, the changes in the fundus may vary from normal to simple or secondary atrophy of the optic nerve or may reveal a choked disk, which may be either unilateral or bilateral. With these usual clinical data available, a definite conclusion as to the type of pathologic involvement still cannot be made, so it is necessary to obtain further help from the neurosurgeon and the roentgenologist. It is well known

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From the Department of Ophthalmology, Temple University School of Medicine.

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that ventriculography renders no aid, but encephalography has been of real service. The roentgenologist usually reports that the pathways of the subarachnoid fluid are somewhat interfered with at the vertex, the cisterna chiasmatica is irregular and deformed and compensatory channels are apparent in the frontal and occipital poles. Slight cortical atrophy is suggested by the enlargement of the pathways around the islands of Reil and the occasional presence of a very large channel between the convolutions over the vertex. These encephalographic findings are strongly against the existence of a space-taking lesion within the cranium. With these additional positive data, as revealed by the encephalogram properly interpreted by the roentgenologist, the ophthalmologist can predicate the underlying pathologic process and advise early neurosurgical therapy.

Three cases are reported in which the correct site of the lesion was diagnosed ophthalmologically and the correct pathologic process predicated preoperatively by the encephalographic findings.

REPORT OF CASES

CASE 1.—A white youth aged 17 was first examined in the Temple University Hospital in June 1935 because of progressive failure of vision in the right eye for three months. At the age of 5 years the patient had severe frontal headaches, which persisted for two days, and these have recurred about every two months. Three years before the present examination he began to grow rapidly and now is 6 feet 2 inches (188 cm.) in height and weighs 158 pounds (72 Kg.). No history of injury or any recent illness could be elicited.

Physical examination gave negative results except for a definite mild acromegalia. Neurologic examination gave negative results.

Ophthalmologic examination revealed the vision to be 6/60 eccentrically in the right eye and 6/6 in the left. The pupils and reflexes were normal; the perimetric fields showed a temporal hemianopia with loss of central fixation for the right eye and normal fields for the left eye. Examination of the fundi revealed nothing abnormal. A diagnosis of a prechiasmal lesion on the right side was made (fig. 1).

The roentgenograms of the head and sella turcica were normal. The encephalogram showed some air in the cisternae at the base, but these were only partially visualized, and there was air in the cisternae chiasmatica.

A right transfrontal exploration was done on June 23 (Gotten) and revealed a definite adherent arachnoiditis of the right frontal lobe, optic nerves and chiasm. These adhesions were freed by blunt dissection, and free spinal fluid escaped. The patient's convalescence was uneventful.

The patient returned four years later because of a nonrelated orthopedic condition. Ophthalmologic examination revealed the vision to be 6/6 in each eye, and the perimetric fields were normal both for form and for colors. The fundi were normal.

CASE 2.—A white girl aged 14 years was referred to the ophthalmic dispensary of Temple University by a school nurse on Nov. 22, 1935 because of blurred vision in the temporal field of the right eye for four weeks. This was first noticed one month previously by the patient and was not accompanied by any discomfort.

Ophthalmologic examination revealed the vision to be 6/10 in each eye. The pupillary reflexes and ocular rotations were normal. There was an acute choked disk in the fundus of the right eye, with elevation of 4 diopters, while the fundus of the left eye was normal. The perimetric fields revealed a right temporal hemianopia for form and colors, with preservation of central fixation. The fields for the left eye were normal. A diagnosis of a prechiasmal lesion on the right side was made (fig. 2).

The patient was admitted to the Temple University Hospital on November 25. General and neurologic examinations gave entirely negative results. The roent-

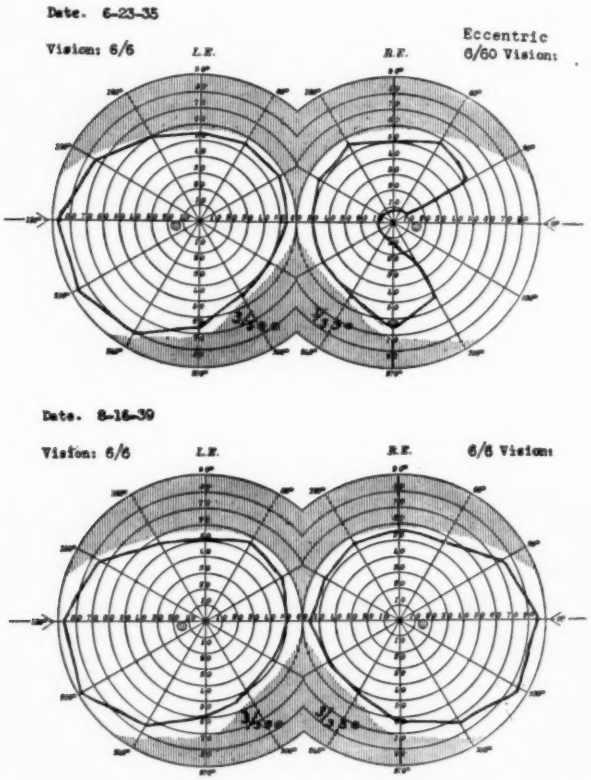

Fig. 1 (case 1).—Right temporal hemianopia with loss of central fixation. The left field of vision is normal. The fundi are normal in both eyes. After operation for basal arachnoiditis, the visual field of the right eye returned to normal.

genograms of the sella turcica and the optic foramen were normal. Encephalographic examination revealed that the pathways of the subarachnoid fluid were somewhat interfered with at the vertex, and compensatory channels were apparent in the frontal and occipital poles. There was no evidence of any space-taking lesion (fig. 3). A right frontal exploration was performed on December 4 (Fay). A definite basal arachnoiditis was found, and a dense, sail-shaped, adherent band involved the mesial aspect of the right optic nerve as it emerged from the optic canal. This was freed by blunt dissection. The convalescence was uneventful.

The patient has been observed regularly since the operation. The edema of the disk has disappeared, but the right disk is somewhat pale and the margins remain blurred. There has been a slight enlargement of the temporal fields, but a distinct hemianopia has persisted. The central visual acuity has remained 6/10.

CASE 3.—A white boy aged 10 years was first examined on Nov. 12, 1936 because of progressive loss of vision in the right eye for five months. His earlier life had been uneventful except for two bicycle accidents, and he had always been

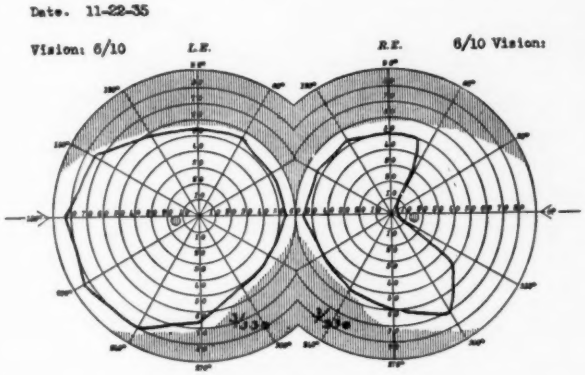

Fig. 2 (case 2).—Right temporal hemianopia with preservation of central fixation. The left field of vision is normal. There was acute choked disk, of 4 D, in the right eye and normal fundus in the left eye.

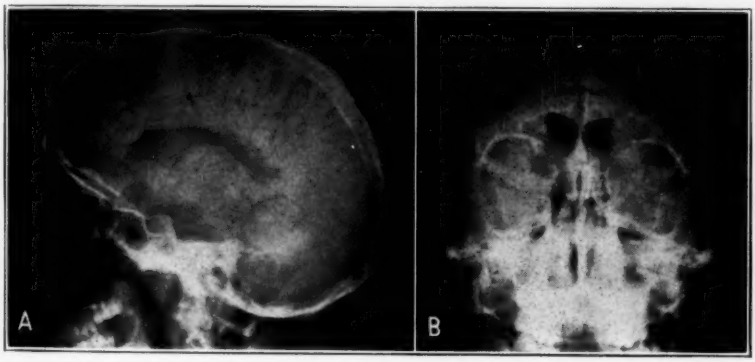

Fig. 3 (case 2).—*A*, lateral encephalogram revealing a normal sella turcica and interference with the pathways of the subarachnoid fluid. *B*, anteroposterior encephalogram revealing dilated cortical subarachnoid channels.

considered as a "problem child." Ophthalmologic examination revealed the vision in the right eye to be limited to ability to count fingers; vision in the left eye was 6/10. The pupillary reflexes were normal, and a definite concomitant convergent strabismus of 5 degrees was present. Examination of the fundi revealed simple atrophy of the optic nerve in the right fundus and a mild pallor of the left disk. The visual fields revealed temporal hemianopia with loss of central

fixation in the right eye and normal fields in the left eye. A diagnosis of a prechiasmal lesion on the right side was made (fig. 4). The patient was sent to the Temple University Hospital on November 12. General and neurologic examinations gave entirely negative results. The roentgenograms of the sella turcica and the optic foramen were normal. The encephalograms showed the basal cisternae to be greatly enlarged, while the pathways of the subarachnoid fluid over the vertex

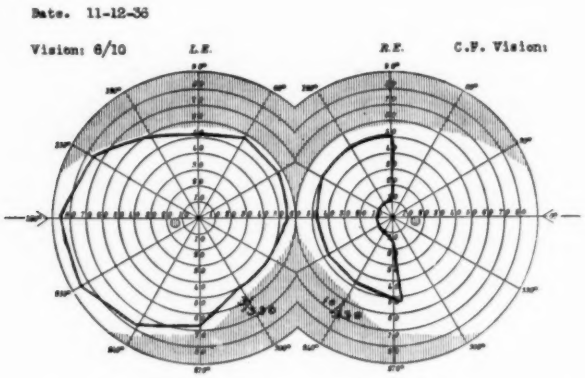

Fig. 4 (case 3).—Right temporal hemianopia with loss of central fixation. The left field of vision is normal. There are simple optic atrophy on the right and pallor of the optic disk on the left.

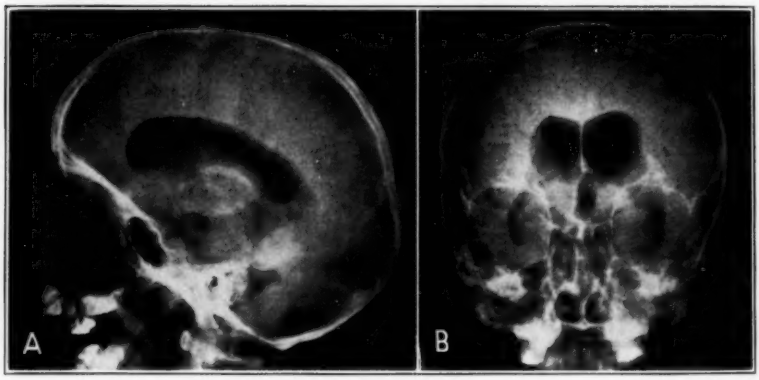

Fig. 5 (case 3).—*A*, lateral encephalogram revealing a normal sella turcica, mild internal hydrocephalus and interference with the subarachnoid channels. *B*, anteroposterior encephalogram revealing dilated ventricles and interference with the cortical subarachnoid pattern.

were either obliterated or incompletely visualized. The cisterna chiasmatica was irregular and deformed (fig. 5).

A right transfrontal exploration was performed on November 20 (Fay), and a widespread thickened and cystic arachnoid was encountered. The right optic nerve was covered with a milky white arachnoid. All adhesions were bluntly dissected

until the chiasm and optic nerves were entirely free. The postoperative convalescence was uneventful. A report from the patient two years later revealed that he is physically and mentally active and that his vision has shown no further deterioration.

COMMENT

In 1931 Craig and I [1] published a paper reporting 8 cases of chronic local arachnoiditis, 7 of which presented the classic ophthalmologic syndrome of a chiasmal lesion and 1 of which presented a definite prechiasmal syndrome. We concluded that chronic local arachnoiditis was a definite clinical entity and produced syndromes similar to tumors in the same region. We also stated that a definite preoperative diagnosis of the underlying lesion could not always be predicated. Vail [2] in 1938 reported a case of optochiasmic arachnoiditis, stressing the importance of a mixed type of atrophy of the optic nerve as a diagnostic sign, although not pathognomonic of it. In all known published reports to the present time a positive diagnosis has been made only at the operation.

The lesion in the 3 cases herein presented was definitely diagnosed as chronic local arachnoiditis before the operation was instituted. This was possible only because of the roentgenographic and encephalographic findings, as the ophthalmologic syndrome could have been produced by any inflammatory, vascular or neoplastic lesion.

CONCLUSIONS

In all cases presenting a definite ophthalmologic syndrome of the prechiasmal or chiasmal type associated with a normal roentgenographic picture, chronic local arachnoiditis should be suspected and encephalographic studies should be made. The latter addition to the diagnostic armamentarium has proved invaluable in predicating the correct pathologic process before surgical treatment is instituted.

Chronic local arachnoiditis responds favorably only to surgical intervention, and an operation should be done without delay.

ABSTRACT OF DISCUSSION

Dr. Temple Fay, Philadelphia: I wish to emphasize the importance of the neuro-ophthalmologist to the neurosurgical clinic for the proper localization and diagnosis of intracranial lesions. Dr. Lillie and the members of his department correctly diagnosed these 3 cases before encephalographic studies confirmed the character of the lesion. He has been extremely modest in claiming credit which should go to him for such refinement in diagnosis.

My associates and I have learned to await the neuro-ophthalmologic report in cases of intracranial lesions before requesting roentgenograms

1. Craig, W. M., and Lillie, W. I.: Chiasmal Syndrome Produced by Chronic Local Arachnoiditis, Arch. Ophth. **5**:558-574 (April) 1931.

2. Vail, D.: Optochiasmic Arachnoiditis, Arch. Ophth. **20**:384-394 (Sept.) 1938.

in view of the greater localizing value it has if positive and the need for more intensive roentgen studies if negative. This is economically important to the patient, as roentgenograms are expensive and a large percentage of them are taken without definite purpose but in the hopes that they may reveal some hidden pathologic process.

The second and third cases presented by Dr. Lillie required courage on the part of the ophthalmologist to urge operation. After encephalography, it was felt that the lesion in the second case was not a matter of life and death, and I was inclined to hesitate because of the surgical risk (every one has due respect for such neurosurgical procedures), but Dr. Lillie urged relief to save vision and pointed out that the risk was less than in the case of tumors. This is true.

In this type of operation the neurosurgeon is faced with three definite requirements: He must safely bring the patient through the operation, because, after all, it is only "sight" that is involved in this problem and not a question of a progressive lesion such as is seen in cases of tumor in which life itself may also be at stake. Not only must the patient be brought through the operation to recovery but he must have a cosmetic result which is commensurate with the undertaking, and the relief of the symptoms must also be established.

(Slides were presented.)

In this slide (Chamberlain's careful technic for encephalography) one can see the chiasmic region clearly outlined, and stereoscopically one can trace these structures so that adhesions come into view and can be actually seen under proper technic.

In this slide the involvement of the basal cisternae is shown, which, associated with the ophthalmologic findings, gives one the diagnosis.

This slide shows the postoperative cosmetic result. Even in a person with a high forehead, the scar is maintained entirely behind the hair line.

As the pituitary region is approached, release of subarachnoid fluid gives a proper exposure of the part, as shown in this slide. The operative field is not hampered by increased pressure, as in tumor, so that the results are better than those in cases of intracranial pressure.

It is possible to elevate the frontal lobe easily after the subarachnoid spaces have drained. One therefore has an opportunity to see not only the chiasm but the structures on the opposite side in this approach and can deal with the adhesions on the side of operation as well as those on the opposite side.

Dr. Derrick Vail, Cincinnati: The presurgical diagnosis of optochiasmic arachnoiditis still is one of utmost difficulty. As Dr. Lillie pointed out, a positive diagnosis can be made only at operation; still, the condition may be suspected from the clinical findings with some degree of confidence. The fact that encephalography played such an important role in the preoperative diagnosis in Dr. Lillie's cases is of great importance, although I do not believe that a normal encephalogram excludes the possibility of arachnoiditis. A positive encephalographic picture, however, adds a heavy weight to the balance of suspicion, and, along with other evidence, such as the history, defects of the visual field, the appearance of the optic nerve head and the absence of signs pointing to other forms of chiasmal involvement, should allow one to make a preoperative diagnosis with a little more confidence.

A second matter of importance which Dr. Lillie has brought out is the addition of these verified cases of this disease to the rapidly growing number already reported. It is important, because there are some who question and dispute the clinical entity here discussed.

This slide (presented at the meeting) shows differential diagnoses of various conditions affecting the chiasm, as tabulated by Walsh. In the last column one sees that this is a condition which occurs from childhood to early adult life. There is a combination of primary or mixed type of atrophy—symmetric defects of the peripheral fields in a sense that they are not synonymous but are symmetric in either the temporal or the nasal side, in other words, the chiasmal field. The pressure symptoms are absent. There are, as a rule, no glandular changes. Roentgenograms reveal no evidence of sellar damage, and finally, there is the additional evidence presented by Dr. Lillie, of the encephalographic findings, which are important.

Dr. W. Ivan Lillie, Philadelphia: I do not want to leave the impression that basal arachnoiditis does respond to medical treatment. It is a surgical condition, and the sooner surgical intervention is instituted, the better the patient's chance for regaining normal or improved vision.

SPARING AND NONSPARING OF "MACULAR" VISION ASSOCIATED WITH OCCIPITAL LOBECTOMY IN MAN

WARD C. HALSTEAD, PH.D.

A. EARL WALKER, M.D.

AND

PAUL C. BUCY, M.D.

CHICAGO

In spite of the detailed comparative anatomic studies of Minkowski,[1] Brouwer and Zeeman,[2] Poliak[3] and others,[4] the macular projection on the cerebral cortex is still a subject of controversy. Lack of satisfactory methods for mapping the central visual fields of animals leaves the functional significance of such anatomic studies in question. On the other hand, there have been few reports thus far of histologic studies of the lesion in human beings combined with controlled investigation of the visual functions.

Neurosurgical advances in recent years have made possible the investigation of patients with circumscribed cerebral lesions under the controlled conditions of the experimental laboratory. During the past three years, we have been privileged to study 2 cases presenting unusual opportunities for a detailed investigation of the cortical projection of the macula.

REPORT OF CASES

CASE 1 (Halstead,[5] case 22).—*Anamnesis.*—M. D., a 25 year old filing clerk, was referred by Dr. M. M. Hoeltgen to the University of Chicago Clinics on Sept.

From the Otho S. A. Sprague Memorial Institute and Division of Psychiatry, and the Division of Neurosurgery, University of Chicago.

Read at a joint meeting of the Section on Ophthalmology and the Section on Nervous and Mental Diseases at the Ninety-First Annual Session of the American Medical Association, New York, June 14, 1940.

1. Minkowski, M.: Zur Physiologie der Sehsphäre, Arch. f. d. ges. Physiol. **141**:171-327, 1911.

2. Brouwer, B., and Zeeman, W. P. C.: The Projection of the Retina in the Primary Optic Neuron in Monkeys, Brain **49**:1-35, 1926.

3. Poliak, S.: Projection of the Retina upon the Cerebral Cortex Based upon Experiments with Monkeys, A. Research Nerv. & Ment. Dis., Proc. (1932) **13**: 535-557, 1934.

4. Walker, A. E., and Fulton, J.: The Thalamus of the Chimpanzee: III. Metathalamus, Normal Structure and Cortical Connections, Brain **61**:250-268, 1938.

5. Halstead, W. C.: Preliminary Analysis of Grouping Behavior in Patients with Cerebral Injury by the Method of Equivalent and Non-Equivalent Stimuli. Am. J. Psychiat. **96**:1-30, 1940.

27, 1937. For ten years she had noticed visual hallucinations occurring every few months and lasting about ten minutes. This disturbance was described as flickering of colored spots directly before her eyes. About a year before admission the attacks were followed by generalized headaches, which lasted several hours. Frequently she vomited with these headaches, but although she felt generally better after the emesis, the headache was not relieved. Just prior to her admission the attacks occurred every few days, and at times the headache was unassociated with the visual phenomena.

Her family and past histories and systemic inquiry revealed no relevant data.

Examinations.—Physical examination showed no abnormalities. The blood pressure was 116 systolic and 68 diastolic. Neurologic examination gave completely negative results except for bilateral choked disks (right, 1 D.; left, 2.5 D.) and a slight increase in the tendon reflexes on the left side. The visual acuity was normal (right eye, 1.0 — 1; left eye, 1.0), and the visual fields were full (fig. 4). Audiometric and vestibular tests gave negative results.

Urinalysis and examinations of the blood, including the Wassermann test, showed no abnormality. Roentgenograms of the skull were interpreted as normal, but subsequent findings led to a review of the films, which showed that the left occipital bone was larger than the right and that the sagittal sinus was deviated to the right in the occipital region. Electroencephalography after attempted ventriculography showed a bilateral absence of alpha waves and slow waves (one-half per second) in the left occipital lead.

Because the neurologic findings were so slight, ventriculography was attempted on September 30, local anesthesia being used. On puncturing the left occipital lobe a cavity containing 15 cc. of clear, lemon yellow fluid was encountered 2 cm. from the surface. Into this cavity 20 to 30 cc. of air was injected. An attempt to puncture the right lateral ventricle was unsuccessful. Roentgenograms taken after this procedure showed a lobulated collection of air in the left parieto-occipital region measuring about 6 cm. in diameter and 3 cm. in width.

Within a few minutes after the patient returned to her room a left homonymous hemianopia was demonstrated. In the course of the following week this hemianopia gradually receded, leaving only a linear scotoma in the horizontal meridian on the left side. The right visual fields showed an inferior partial quadrantic defect, which did not change much until the time of her second operation. Because a cold developed, this procedure was not carried out until October 23.

Operation and Course.—Anesthesia was produced with avertin with amylene hydrate and local infiltration of the scalp, and a left osteoplastic craniotomy was performed. The occipital lobe was yellow and its gyri flattened. An incision was made parallel to and slightly posterior to the external parieto-occipital sulcus and carried through the white matter anterior to the tumor and cyst. The anterior margin of the amputation was 1 cm. posterior to the edge of the tentorium cerebelli in the midline.

The convalescence was smooth. Immediately on awakening from the anesthetic the patient talked coherently and read without hesitation. At no time was there any evidence of aphasia or alexia. She had complete right hemianopia, and frequently misplaced objects in her blind field. She was discharged on November 11.

Since leaving the hospital, the patient has been seen on numerous occasions. Her neurologic status has remained normal except for the right homonymous hemianopia. Her fundi are normal. She continues to have hallucinations of flashing colors and lights. Her visual acuity since operation has been (with correction) 0.8 in the right eye and 0.8 + 2 in the left eye.

Anatomic Report.—The operative specimen consisted of a block of tissue, which could be readily recognized as the left occipital lobe. It measured 7 cm. antero-posteriorly along the sagittal sinus and 9.5 cm. along the lateral surface of the lobe. The parieto-occipital fissure could not be positively identified. Three and five-tenths centimeters posterior to the line of ablation, a reddish brown mass occupied the entire occipital pole. It extended 4 cm. anteriorly from the posterior tip of the occipital lobe and 3.5 cm. laterally from the midline. On the medial surface of the lobe the calcarine fissure was seen to extend from the superior margin of the tumor anteriorly to the incision. An obliquely horizontal section made through the tumor and occipital lobe revealed a cystic cavity about the anterior, inferior and lateral margins of the tumor. This cavity measured 5 cm. antero-posteriorly and 3.5 cm. in height and extended 1.5 cm. anterior to the tumor (fig. 1).

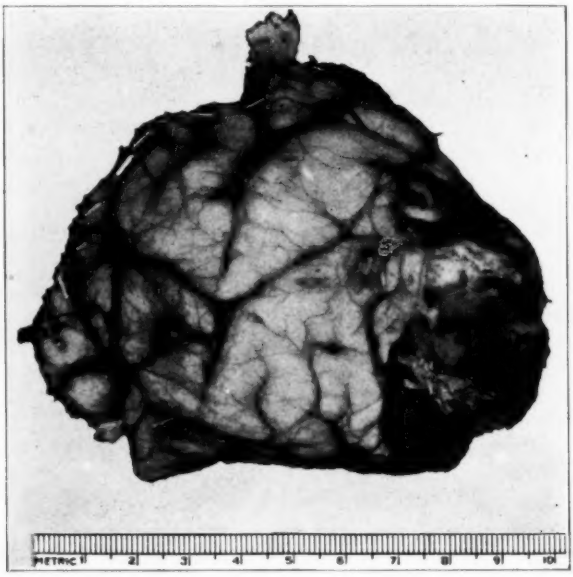

Fig. 1 (case 1).—Photograph of the lateral surface of the ablated tissue.

The two blocks were replaced in their normal anatomic relationship, dehydrated and embedded in pyroxylin. Serial coronal sections were made at 20 microns, every twelfth section being saved. Of these, every other one was stained for myelin, every fourth by Nissl's technic and the remainder with hematoxylin and eosin.

After studying the sections in detail, approximately every two-hundredth was drawn with the aid of an Edinger projector at a magnification of 2. The details were filled in by microscopic control. In figure 2 the striate cortex is indicated by heavy stippling and the tumor by black.

Examination of the serial sections showed that the tumor, although separating the lips of the calcarine fissure posteriorly and displacing the striate cortex superiorly, had actually invaded the striate cortex very little. The operative trauma

and distortion of the tissue due to fixation had so distorted the cortex of the occipital pole that it was difficult to be certain of the orientation of certain parts. It was obvious, however, that little or none of the striate cortex had actually been invaded by tumor. The tumor mass extended forward to about the anterior third of the striate cortex, but the cyst, the wall of which was composed of tumor tissue, reached almost to the anterior level of the striate cortex. The latter could be followed on the lips of the calcarine fissure, more on the superior than on the inferior lip, almost to the rostral portion of the ablation. There were, however, several millimeters of area 18 cortex in the depth of the calcarine fissure rostral to the striate area.

The tumor was a mixed astrocytoma and polar spongioblastoma.

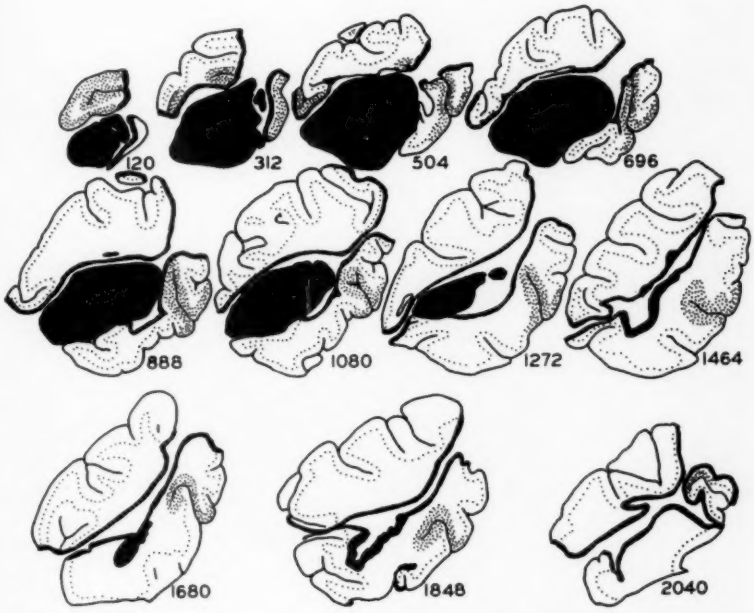

Fig. 2 (case 1).—Serial sections of the ablated occipital lobe, the most posterior section being above and to the left and the most anterior to the right and below. The tumor is indicated by solid black and the striate cortex by stippling.

Summary: The ablation had removed completely the left striate cortex and areas 18 and 19 of the occipital lobe posterior to the parieto-occipital fissure.

Analysis of the Central Visual Fields.—In analyzing the central visual fields use has been made of (1) a standard test room, (2) a projection campimeter and (3) an electrical method of recording ocular movements occurring during fixation. The test room consists of a room 5 by 7 by 6 feet (152 by 213 by 183 cm.), located inside a laboratory room which has controlled illumination. The room is electrically shielded, partially sound-proofed, light-sealed and ventilated. In a door at one end of the test room is mounted a section of milk glass which serves as a campimeter screen. A specially constructed chair, equipped with a chin rest and a head holder, orients the visual axis of the subject with reference to the center of the screen. With the subject thus oriented, the central visual field may be

mapped by means of targets projected onto the screen by lanterns in the outer laboratory room (fig. 3).

The electrical method employed for recording ocular movements occurring during fixation has been described by one of us (Halstead,[6] 1938). The method involves the detection, amplification by a vacuum tube amplifier and recording on wax-coated paper or on bromide paper of changes in the corneoretinal potential associated with movements of the eyes. A small silver electrode applied to the skin behind each of the two orbits plus a grounding electrode are the only necessary connections to the subject. The method offers the advantages that with appropriate amplification ocular movements of less than 30 minutes of arc may be detected and that it imposes no mechanical obstruction of the visual field.

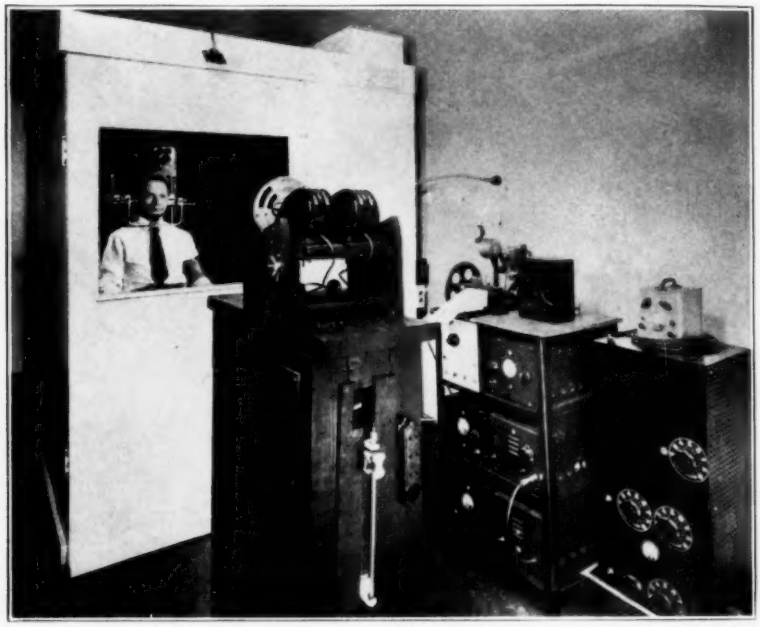

Fig. 3.—Shielded perimetric room with the head rests from which the milk-glass window has been removed (left), the projecting apparatus (center) and the equipment for recording corneoretinal potentials (right).

Discrimination of Brightness and Color: The results of charting the central fields of this patient with simultaneous control of fixation by the method described are shown in figures 4 and 5. The concentric circles shown in figure 5 represent intervals of 1 degree of visual angle. It may be seen that for the right eye sparing of central vision for brightness of 2.5 degrees was found; for the left eye sparing of approximately 2 degrees was found. Sparing of central vision for color was found to correspond to that for brightness in both eyes. (In mapping for color use was made of Wratten monochromatic filters nos. 70, 71A, 72, 73, 74 and 75.)

6. Halstead, W. C.: A Method for the Quantitative Recording of Eye Movements, J. Psychol. **6**:177-180, 1938.

Discrimination of Form: Under conditions of the standard test room, targets differing only as to shape or form were projected tachistoscopically into various parts of the central field. The targets employed consisted of a square, a circle and a triangle, each subtending 15 minutes of visual angle. The central field was explored through 3 degrees at intervals of 20 minutes of arc. Control exposures

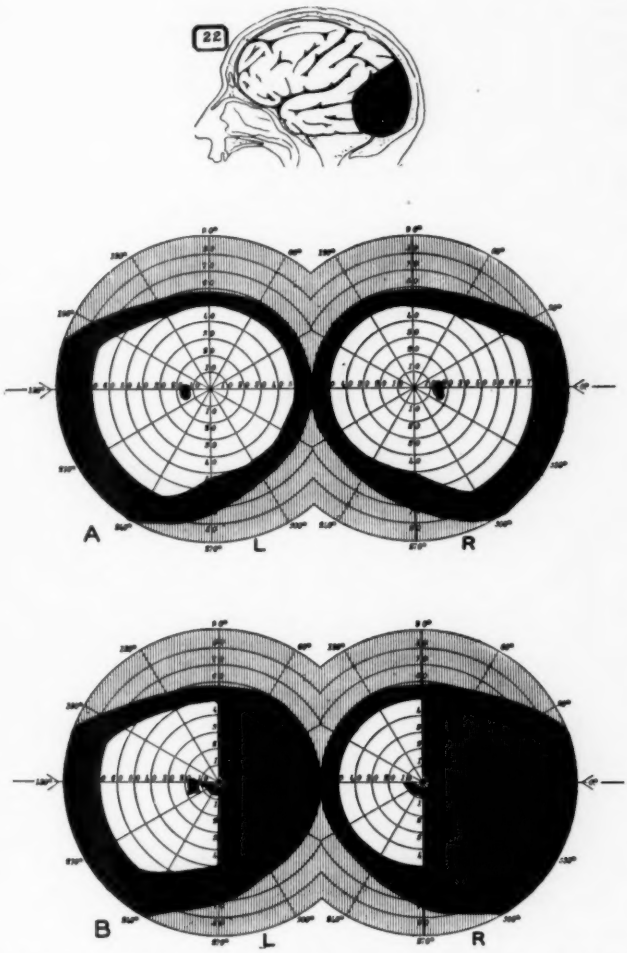

Fig. 4 (case 1).—The lesion (above), the preoperative visual fields (*A*) and the postoperative visual fields (*B*). In all illustrations the field for the right eye is to the right and that for the left to the left.

were presented in two ways: (1) on corresponding points in the sense of being symmetrically located with reference to the fixation point of the intact field and (2) without exposure of the target. An exposure interval of one-fiftieth second was found to give a maximum field. This interval is approximately one-fifth that required for an ocular movement.

As may be seen in figure 5, the spared field in terms of form discrimination was found to be 1.25 degrees maximally, or approximately half that found to be spared for brightness and color. It is noteworthy that the spared region is noncongruous in the two eyes.

Findings likewise indicative of true sparing of central vision have been obtained under additional test situations.[7]

1. During the sixth postoperative week, the central field was mapped on a specially prepared campimeter, while continuous photographic records of horizontal and vertical ocular movements and slight head movements were simultaneously obtained.[8] The central field for brightness was charted for the right eye at distances of 50 cm. and 1 meter. A 2 mm. flat white target was used against a flat black cardboard screen. The vocal signal of the patient was recorded on the film by means of a microphone pick-up. In figure 6 the photographic records

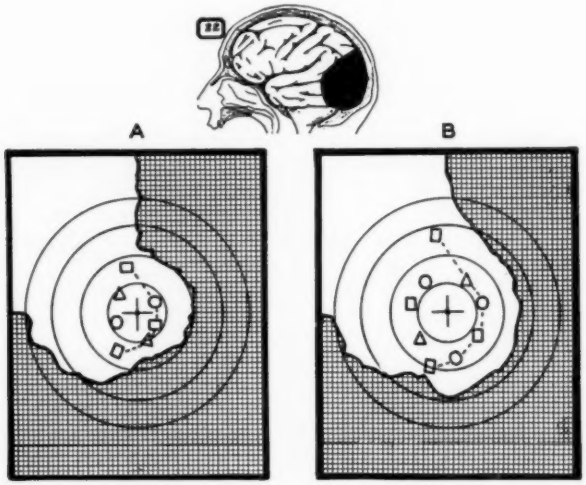

Fig. 5 (case 1).—The central visual fields for brightness and color (solid line) and form discrimination (dotted line) after left occipital lobectomy.

associated with a point mapped in the central field are reproduced. The point considered here represents the peripheral boundary of the central field on the hemianopic side. It was found to be stable at 2.5 degrees.

2. Repeated tests on the Bjerrum screen by four different examiners in every instance yielded evidence of sparing of approximately 4 degrees in each eye.

3. Repeated tests on the Lloyd stereocampimeter by two different examiners likewise revealed sparing of approximately 3 degrees in each eye.

4. Repeated tests on a projection campimeter under photopic and scotopic conditions, using an artificial pupil with reflected and projected targets, yielded maximal sparing of 2.5 degrees.

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7. A description of the various additional methods employed and a discussion of their relative accuracy is being prepared for publication by one of us (Halstead).

8. Prof. Guy Buswell cooperated by securing these records by means of the University of Chicago eye movement camera.

5. Repeated tests on a flicker campimeter, using targets presented at various flash frequencies, revealed sparing of approximately 2.5 degrees.

6. Repeated perimetric examinations, using a modified Ferree-Rand perimeter, revealed sparing of from 4 to 5 degrees.

7. Repeated tests by two examiners on the Zeiss perimeter, using projected targets, yielded sparing of 3.5 degrees.

8. Reliable recognition of form when presented on a projection campimeter behind the fixation point and then moved into the blind field, with direct corneo-retinal control of fixation, was elicited.

9. Repeated tests for the minimum exposure interval for recognition of geometric forms revealed evidence of acuity in the spared region equal to that of the intact field.

10. Repeated tests demonstrated the functional availability of the spared area for the perception of apparent movement (Phi effect) with reliable boundaries.

Certain additional lines of evidence supporting the foregoing evidence have been derived from various control investigations. These include:

1. Subjective control of fixation by the patient in terms of colored light of high intensity projected onto her blindspot was demonstrated. When seen, the colored light reveals to the subject the fact that fixation has been disturbed. It was found that this patient could fixate and maintain fixation with a high degree of accuracy.

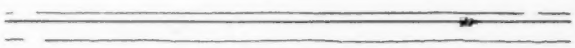

Fig. 6 (case 1).—Photographic record of head movement (lower line), microphonic pick-up of vocal signal (middle line) and lateral ocular movement (upper line) during the presentation of a test object. The slightest movement of head or eyes produces a hump in the line. The discontinuity of the lines at the left indicates the introduction of the test object into the visual field. There is no head or ocular movement until after the recognition of the stimulus when an ocular movement is seen. The speed of the film was such that ½ inch (1.3 cm.) on the illustration is the equivalent of 1 second.

2. It was found that negative after-images developed normally on the spared portion of the central field.

3. Investigation revealed systematic constancy of distance between the peripheral spared boundary and the nasal boundary of the blindspot.

4. Investigation revealed systematic constancy of amount of sparing as a function of mapping distance.

5. Investigation revealed evidence of stability and systematic constancy of the paramacular scotoma as a function of mapping distance.

6. Investigation by the localization and recognition methods of Fuchs [9] demonstrated absence of pseudofovea.

Summary.—An intelligent, cooperative patient from whom all of the left striate cortex had been removed, as shown by histologic examination

9. Fuchs, W.: Eine Pseudofovea bei Hemianopikern, Psychol. Forsch. **1:** 157-186, 1922.

of the operative specimen, was found to have sparing of central vision for color and brightness of 2.5 degrees maximally and sparing for form of 1.25 degrees maximally.

Comment.—The left homonymous hemianopia, which developed immediately after the attempted ventricular puncture, was probably the result of the passage of the needle near the trough of the right calcarine fissure. This accident resulted from the fact that the sagittal sinus was displaced to the right side, so that the right burr hole, although correctly placed in relation to the skull, was actually just to the side of the sinus. The subsequent disappearance of this hemianopia, leaving but a small paracentral scotoma, indicates that the greater part of the involvement of the striate cortex was probably the result of circulatory disturbances and edema.

The first examination of the central fields was made on the eighth postoperative day. With the exception that the boundaries obtained have become more reliable, there has been little change in the fields during the past three years.

CASE 2 (Halstead,[5] case 25).—*Anamnesis.*—I. M., a 22 year old stenographer, was admitted to the University of Chicago Clinics on Sept. 27, 1939, because of right-sided headaches. About three years previous to admission she began to have headaches beginning above the right eye and radiating to the right occiput. Associated with these headaches were usually yellow and green scotoma-like specks, which floated into space. The patient did not refer these visual hallucinations to any particular part of the visual field. The headaches occurred in the late morning, afternoon or evening and lasted for several hours. Initially the headaches occurred about once a week, but for a few months before the patient entered the hospital they increased in frequency and severity and were associated with nausea. To relieve this, she induced vomiting and found that it also relieved her headache.

Her family and past histories and inquiry by systems yielded no further relevant data.

Examinations.—Physical examination revealed a pleasant young girl in no obvious distress. No abnormalities were detected in the heart, lungs or abdomen. The blood pressure was 128 systolic and 76 diastolic. Neurologic examination also gave negative results except for left homonymous hemianopia. Visual acuity was 0.8 + 3 for the right eye and 1.2 — 2 for the left eye. The patient was right handed. With the revised Stanford Binet test (L) she rated an intelligence quotient of 133; with the Kuhlmann test, which makes greater demands on speed of performance, the intelligence quotient was 97.

Urinalysis and examinations of the blood showed no abnormalities. A lumbar puncture was made. The initial pressure was 125 mm. of spinal fluid, and a good rise was obtained on compression of the jugular vein. The fluid was clear and colorless and contained 2 lymphocytes per cubic millimeter. The total protein was 40 mg. per hundred cubic centimeters and the Lange curve 0011000000. The Wassermann test with both the blood and the spinal fluid was negative.

Roentgenograms of the skull showed a calcification in the right occipital region, with an area of thinning of the right occipital bone. An electroencephalogram showed a striking difference in the alpha waves from the occipital leads,

those on the right side being very small (Case [10]). Questionable delta waves were present in the right occipital region.

The location of the lesion was obvious from the clinical, roentgenographic and electroencephalographic examinations, but its nature was not so clear, although neoplasm seemed likely. For that reason encephalography was attempted, but the ventricular system did not fill and there was no air over the right cerebral hemisphere although considerable in the sulci over the left. On the following day, September 30, bilateral perforations were made in the occipital region, local anesthesia being used. The dura mater was opened on both sides, exposing normal cerebral cortex on the left but grayish semigelatinous tissue on the right. Several fragments of the latter were removed for biopsy. When puncture of the ventricles was attempted, a cyst was encountered on the right side at a depth of 4 cm., from which olive-colored fluid was obtained. Air was injected into the cyst and promptly appeared from the opposite needle. After 60 cc. of air was passed from one side to the other, the wounds were closed.

Roentgenograms taken after this procedure showed anterior displacement of the trigon of the right lateral ventricle and an air-filled cyst lying just lateral to the calcification in the right occipital lobe.

The patient complained of considerable headache following this procedure. Her visual fields, however, were found to be practically full, and the patient noted that her vision had improved.

On October 10, after anesthetizing the patient with ether, a right posteriorly placed osteoplastic craniotomy was done. After the dura was opened, the occipital lobe was amputated by an incision just posterior to the external parieto-occipital sulcus and the point where the anastomatic vein of Labbe enters the tentorium cerebelli. The line of the incision passed through the posterior horn of the lateral ventricle. After the ablation, the splenium of the corpus callosum was not visualized, but the medial margin of the ablation was 5 mm. posterior to the junction of the inferior longitudinal and straight sinuses.

The patient had an uneventful convalescence. She had no visual distortion or disorientation except for a few minutes immediately on recovering from the anesthetic, when she recalled that one of the doctors appeared unusually tall and thin. At no time had she any aphasic or alexic difficulties. Except for the hemianopia, she had no neurologic abnormality. She was discharged on November 3. Since then she has returned to work. On several occasions she has been seen in the outpatient department and has had no complaints except that the paroxysmal visual hallucinations still occur. Her visual acuity since operation has been 0.6 —2 in the right eye and 0.8 in the left eye.

Anatomic Report.—The operative specimen consisted of a large block of tissue weighing 113 Gm., at one end of which was necrotic, hemorrhagic and soft tumor tissue. At the other end was a rather ragged surgical line of incision through what appeared to be normal gray and white matter. The other three sides of the specimen presented normal-appearing cortex covered by arachnoidal membranes. The specimen measured 7.7 cm. along the superomedial margin, 6.5 cm. along the inferolateral margin and 7 cm. at its widest point. The tumor presented on the surface 2 cm. posterior to the ablation superiorly and 4.5 cm. posterior to the ablation inferiorly. The anterior limit of the ablation was difficult to determine, but it seemed to be just posterior to the external parieto-occipital sulcus. On the medial surface of the hemisphere the calcarine fissure was visible for 2 cm., at which point the sulcus has been cut across (fig. 7).

10. Case, T. J.: An Association Between Homonymous Hemianopsia and Unilateral Absence of the Alpha Waves, Arch. Neurol. & Psychiat., to be published.

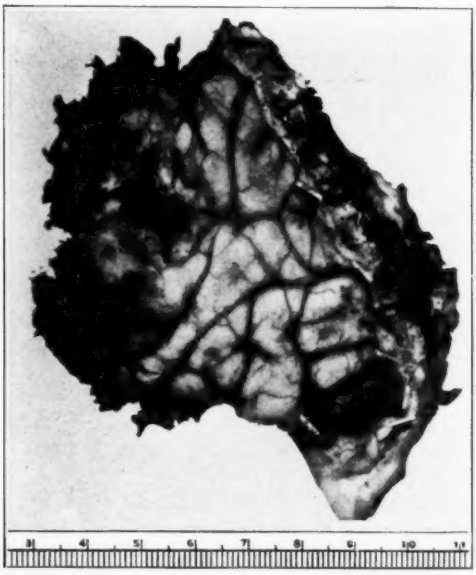

Fig. 7 (case 2).—Photograph of the lateral surface of the ablated occipital lobe.

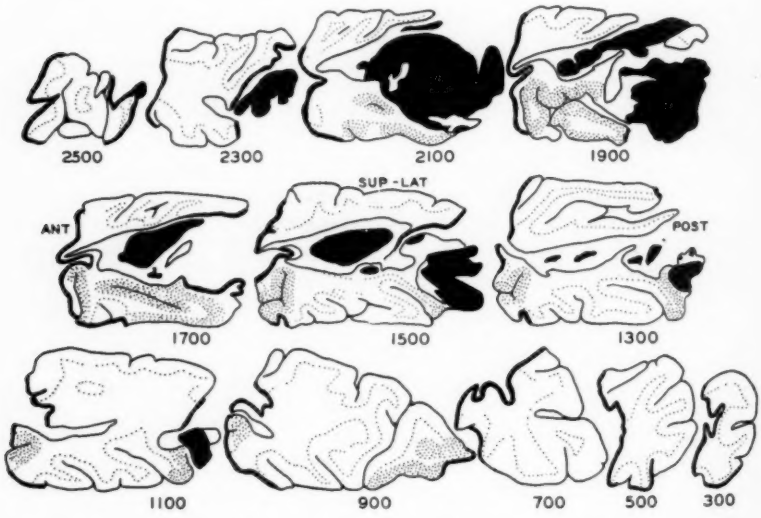

Fig. 8 (case 2).—Serial sections of the ablated occipital lobe cut in a semi-horizontal plane. The tumor is indicated by solid black and the striate cortex by stippling.

The entire block was embedded and sectioned serially, every fifth section being saved. Every twenty-fifth section was stained for myelin by Weil's method and the adjacent one by Nissl's technic. Representative sections of the tumor were stained by hematoxylin and eosin. The block was sectioned approximately at right angles to the convexity of the brain in an almost horizontal plane (fig. 8).

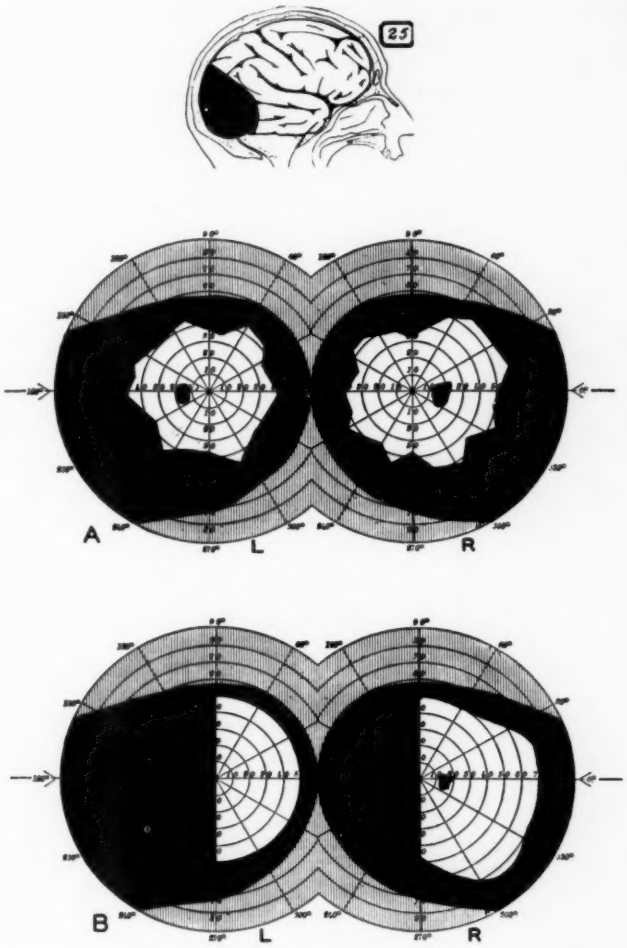

Fig. 9 (case 2).—The lesion (above), the preoperative visual field (*A*) and the postoperative visual field (*B*).

Examination of these serial sections showed the ablation to extend anterior to the tumor, which appeared to be almost surrounded by a gliosed cyst wall. The tumor and cyst lay above the striate cortex, which could be readily distinguished. The plane of section being parallel to the calcarine fissure, the latter was seen on the inferior surface of the section extending from one end to the other (section 1900). There was no doubt that the entire posterior part of the striate

cortex had been removed, but the anterior extremity of the ablated block adjoined striate cortex, and hence it was impossible to state that the entire cortex of area 17 was removed. However, if some was left it must have been little, for in only about 300 sections (6 mm.) did the ablation border striate cortex. In all other sections peristriate cortex was present along the line of ablation. That little of the striate cortex could be left was evidenced by the fact that the calcarine fissure anteriorly became very shallow in the ablated block (sections 1100 and 900). Since the striate cortex does not extend to the anterior end of the calcarine fissure it is probable that the ablation contained almost all of the striate cortex. The tumor was a typical oligodendroglioma.

Analysis of the Central Visual Fields.—In analyzing the central visual fields for brightness and color the test conditions described in experiment 1 were employed.

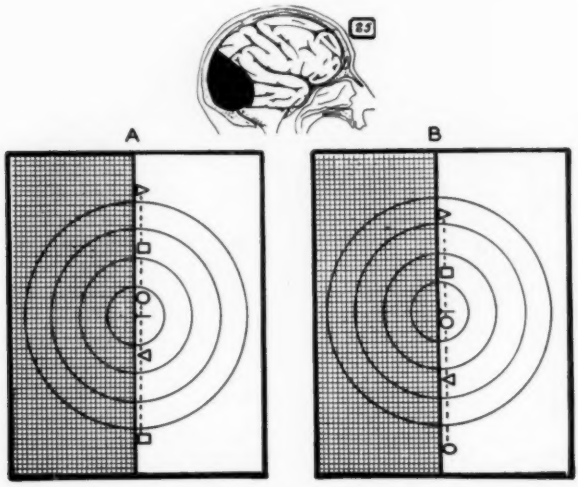

Fig. 10 (case 2).—The central visual fields for brightness and color (solid line) and form discrimination (dotted line) after right occipital lobectomy.

These included (1) a standard test room, (2) a projection campimeter and (3) an electrical method of controlling movements of the eye during fixation.

Discrimination of Brightness and Color: The results of charting the central fields of this patient with simultaneous control of fixation during the tenth post-operative week are shown in figures 9 and 10. The concentric circles shown in the figure represent intervals of 1 degree of visual angle. It may be seen that no evidence of sparing of central vision for brightness was found for either eye. It was found that there was likewise no evidence of sparing for color in either eye (the boundary obtained conformed to that for brightness).

Discrimination of Form: Tests for discrimination of form were made under conditions similar to those described in experiment 1. As may be seen in figure 10, the limits of form vision were found to correspond to those for brightness and color and to pass through the center of the fixation point in each instance. It should be noted that some adjustment of this vertical boundary toward the intact field was necessary to correct for completion effect discovered in the responses

of this patient. The amount of adjustment required was slightly less than 10 minutes of visual angle.

With two exceptions, the additional tests and control investigations described in case 1 were made. The perimetric fields taken on the Bjerrum screen were inconsistent. At times an apparent sparing of 2 degrees in each eye appeared to be present but could not be confirmed by the same or different observers later on the same or another day. Similarly the findings based on the Lloyd stereocampimeter proved unreliable with reference to the splitting or sparing of the central fields.

A second intelligence test (Stanford-Binet test M) made one month after the operation gave a rating of 133.

Summary.—Following ablation of the right occipital lobe, including almost, if not all, of the striate cortex, a splitting of the macula was found on controlled campimetric examinations.

Comment.—This case, unlike the first, is slightly marred by the fact that the entire striate cortex cannot be demonstrated in the serial sections of the specimen. However, since the patient has splitting of macular vision rather than sparing, this possible anatomic defect does not vitiate the interpretation. We believe that the inconsistencies in the central visual fields when determined on the Bjerrum screen or the Lloyd stereocampimeter, examinations which in this case could not be controlled for ocular movements as in case 1 because of technical difficulties, are the result of the defects inherent in the perimetric method. The fields were consistent when determined by the other technics mentioned previously.

COMMENT

In the 2 cases of occipital lobectomy considered in this report it was found that the patients remained intelligent, cooperative and capable of accurate fixation, thus making possible careful examination of the central visual fields. Right hemianopia with noncongruous maximal sparing of 2.5 degrees for brightness and color was found under test conditions, providing direct control of accuracy of fixation in the instance of the patient with the left occipital lobectomy. Sparing of 1.25 degrees for form was also found in this patient. Left homonymous hemianopia with splitting of the macula was found in the instance of the patient with a right occipital lobectomy. This has been found repeatedly on examinations made during the six months after the operation. In the first case complete removal of all striate cortex has been verified histologically. In the second little, if any, striate cortex remained after lobectomy. Only the most anterior extremity of area 17 could have been left.

The paradox presented by the foregoing cases does not seem open to solution in terms of any of the present hypotheses concerning the arrangement of the visual system. The theory of a callosal geniculo-

calcarine bundle, propounded by Pfeifer [11] and elaborated by Foerster [12] and by Penfield, Evans and MacMillan [13] has found no corroboration in comparative neurophysiologic (Maison, Settlage and Grether [14]) or neuroanatomic investigations (German and Brody,[15] Putnam [16] and Poliak [3]) and no support from the recent clinical study of Hyndman.[17] The theory of aberrant macular fibers in the optic chiasm has been questioned by numerous investigators particularly on the basis of comparative material and has received no attention from recent workers. The possibility of retinal internuncial overlap in the macular region is not precluded by investigative studies, but no definite evidence has been produced in its favor. We realize that these theories do not explain the paradox presented. Yet we believe that the evidence afforded by our 2 cases is sufficiently well controlled both in terms of histologic control of the lesion and in analysis of the visual functions to show that the paradox is real and not apparent. The problem of the cortical projection of the macula is not yet adequately solved.

ABSTRACT OF DISCUSSION

DR. WILDER G. PENFIELD, Montreal, Canada: The essayists have presented careful, scientific, controlled evidence that a lesion of the occipital lobe can give rise to hemianopia with a sparing of macular vision. They also reported a case in which there is no sparing of macular vision. From an anatomic consideration, as they have pointed out, there is no explanation for hemianopia with macular sparing. This is not the first time that clinical evidence and anatomic and physiologic evidence

11. Pfeifer, R. A.: Myelogenetisch-anatomische Untersuchungen über den zentralen Abschnitt der Sehleitung, in Monographien aus der Gesamtgebiete der Neurologie und Psychiatrie, Berlin, Julius Springer, 1925, no. 43, p. 2.

12. Foerster, O.: Beitrag zur Pathophysiologie der Sehbahn und der Sehsphäre, J. f. Psychol. u. Neurol. **39**:463-485, 1929.

13. Penfield, W. G.; Evans, J. P., and MacMillan, J. A.: Visual Pathways in Man with Particular Reference to Macular Representation, Arch. Neurol. & Psychiat. **33**:816-834 (April) 1935.

14. Maison, G. L.; Settlage, P., and Grether, W. F.: Experimental Study of Macular Representation in the Monkey, Arch. Neurol. & Psychiat. **40**:981-984 (Nov.) 1938.

15. German, W. J., and Brody, B. S.: The External Geniculate Bodies: Degeneration Studies Following Occipital Lobectomy, Arch. Neurol. & Psychiat. **43**:997-1003 (May) 1940.

16. Putnam, T. J.: Studies on the Central Visual System: II. A Comparative Study of the Form of the Geniculostriate Visual System of Mammals, Arch. Neurol. & Psychiat. **16**:285-300 (Sept.) 1926; III. The General Relationships Between the External Geniculate Body, Optic Radiation and Visual Cortex in Man: Report of Two Cases, ibid. **16**:566-596 (Nov.) 1926.

17. Hyndman, O. R.: The Central Visual System, Arch. Neurol. & Psychiat. **42**:735-742 (Oct.) 1939.

have shown discrepancies. For a long time clinical evidence pointed to motor representation in the cortex before it was discovered physiologically.

In this case, clinically, one knows that lesions of the temporal lobe which interfere with the geniculostriate bundle produce hemianopia which is complete, with macular loss. A lesion of the optic tract gives the same result. A lesion of the occipital lobe may result in macular sparing.

I have observed many cases in which there was no tumor but an atrophic lesion of the brain in which removals could be checked carefully, more carefully than in the case of the tumor, even, and in all of them the lesion of the temporal lobe caused macular loss. Lesions of the occipital lobe, if they did not extend too far laterally or too far forward into the temporal lesion, cause macular sparing.

Poliak in his chimpanzees finds only crossed macular representation in the occipital lobe. Holmes and Lister during the last war presented a case in which operation showed involvement of both occipital poles by a gunshot wound. A central scotoma and complete loss of central vision with the preservation of peripheral vision resulted. This would mean that there is no macular representation anterior to the pole; therefore, removal of one pole could only result in sparing of macular vision if there is a bilateral representation.

So one is faced with the necessity of either disbelieving certain evidence or assuming that there is more evidence to come. Fulton, in his book on physiology, has elected to assume that Holmes and Lister's observation was faulty, because it was carried out in a base hospital during the last war; I may say during the relatively leisurely conditions of the last war.

Those who have worked with Gordon Holmes I think would not agree that he could be mistaken in the examination of a central scotoma. The dilemma can be solved only by assuming that there is a crossing track carrying macular representation to the opposite occipital pole or by assuming that there is a representation of macular vision quite far anteriorly in the same side. The evidence against such a track was first Putnam's evidence, in which he seemed to disprove the track as shown by Pfeifer. Putnam's evidence is carried out by studies on infants. Poliak's evidence was obtained from work on chimpanzees. An anatomic track is needed in the adult, and it seems likely that such a track is subserved, or could be subserved, by collateral branches rather than by necessarily essential axons.

At all events, from a clinical point of view, it does hold that a macular-splitting hemianopia points to a lesion of the temporal lobe or to a lesion anterior to that. A macular-sparing hemianopia points to a lesion of the occipital lobe.

One might suggest that in the case presented today in which there was loss of macular vision the removal or the disturbance of the tumor extended far enough forward so as to constitute a case of interference in the temporal lobe as well as in the occipital lobe. I should like to ask Dr. Walker if he thinks that this explanation might bring the two apparently opposite bits of evidence into line.

DR. P. J. LEINFELDER, Iowa City: The essayists present 2 cases of occipital lobectomy in which the paradoxic condition of sparing and non-

sparing of the macula is well illustrated. In unusually well controlled clinical experimentation, the degree of anatomic disturbance has been carefully determined in 2 cases, and the two different effects in the macular field are illustrated. The writers have been particularly meticulous in the perimetric studies of their patients after lobectomy.

The care and precision utilized in various determinations of the visual field preclude the possibility of unsteady fixation and definitely establish the reliability of the diagnosis. The authors recognize that they have been unable to contribute to a better understanding of the anatomy and physiology of macular sparing, but they have presented additional proof for the clinical reality of the sign. It still remains for the anatomist and the physiologist to explain the how and why of macular sparing.

I should like to emphasize the fact that there is no definite relation between macular sparing and complete or incomplete removal of the occipital cortex. In some events there is a true homonymous hemianopia; in other cases there is a slight bulge of the dividing line toward the blind field, and in additional cases there is a relatively semicircular bulge of 2 to 10 degrees in the central region. In the absence of a macular semicircle, it is difficult to know with certainty whether or not macular sparing is present. The German authors feel that a 2 degree semicircle should be present in order to diagnose macular sparing. The authors' case suits the requirements. The fact that macular sparing occurs in other than lesions of the occipital lobe is not commonly recognized. Wilbrant and Henschen recognized the sign in lesions of the optic tract, and several observers have demonstrated it in disease of the chiasma. In reviewing my own cases of macular sparing, I found a right homonymous hemianopia with 5 degrees of macular sparing in a boy of 16. This had developed following the removal of a cyst from the lateral portion of the occipital lobe. Three months after operation the visual fields were normal. In this case it is probable that edema and hemorrhage caused sufficient pressure on the optic radiations to interfere with conduction. With healing, the pressure was relieved and the integrity of the visual pathway to the cortex was reestablished.

In spite of controversial anatomic studies and the inadequacy of theories of explanation, the fact remains that macular sparing does exist in certain cases of hemianopia. That some hemianopias do not show the sign adds to the confusion, yet it is wise to continue to study the facts and to extend experimental investigation in the hope that the true cause of this interesting condition may be established.

DR. TRACY J. PUTNAM, New York: The few cases which have been presented have been studied probably more carefully than any cases of hemianopia heretofore reported. They set an example of careful perimetric study which I hope will be followed in the future. In a recent review of the literature, since Bunge first suggested that there was a bilateral representation in the macula, it was striking to see that the weak point in practically all presentations on the subject is the perimetric study. It seems to me that for the first time irrefutable evidence is presented that complete destruction of the striate cortex may or may not still leave a remnant of central vision.

Cases of both types have been presented before, and cases have been reported in which a similar sparing of central vision resulted from an extensive resection of the occipital lobe plus a section of the corpus

callosum, which, it seems to me, is substantial evidence that there is no pathway from one geniculate body to the opposite cortex.

The positive anatomic evidence in favor of such a pathway is meager. It rests on the work of Pfeifer, who in a later comment on his own work states that he did not intend to imply that he believed there was such a pathway but merely that if there was such a pathway, perhaps this was the point at which it came off.

In repeating his first work on identical material and with, I believe, more accurate reconstruction, I traced the pathway he described and found it ended in the homolateral cortex. I will not go into the other anatomic studies on this problem. Dr. Walker is aware of them and has summarized them adequately.

One is faced with a number of paradoxes. The first one is that at times apparently the same anatomic lesion leaves a little central vision and at other times it does not. Another paradox is that hemimacular scotomas have been described as extending actually up to the fixation point, apparently from lesions at the tip of the occipital lobe. Autopsy has never been performed in such a case, as far as I know, except the original case of Henschen and Wilbrand, which is unsatisfactory from several points of view.

Dr. W. Ivan Lillie, Philadelphia: The controversy over this question of sparing and nonsparing of the macula can be easily explained without having to think of a separate pathway or bilateral macular representation. It is known from the clinical standpoint that vascular lesions rather than neoplastic lesions or calcified hematomas support the work of Holmes and Lister, who showed that a homonymous central scotoma is obtained if the macular area in the pole of the occipital lobe is involved. This is possible only when the occipital visual area or the optic chiasm is involved. There has never been a case reported in which a homonymous scotoma has been found as a result of a lesion in the temporal lobe or in the optic tract. The central scotomas resulting from lesions involving the optic chiasm are asymmetric; usually one scotoma is much more advanced than the other, and the central visual acuity is markedly reduced. The homonymous central scotomas resulting from a lesion of the occipital lobe do not affect the central visual acuity and are symmetric.

I feel that the present knowledge of the anatomy of the visual area in the occipital region can fully account for the different types of hemianopic defects that one finds clinically; namely, if the occipital pole is spared, there will still be macular preservation. If the occipital pole plus the rest of the visual perception center is involved, a sharp cut of hemianopia is found.

Bilateral representation of the macula is not necessary to correlate the homonymous hemianopias with or without macular sparing produced by lesions of the occipital lobe.

Dr. A. Earl Walker, Chicago: The discussion which this paper has evoked has fully justified our opening statement that this subject is one of great interest and one which lacks complete agreement.

Dr. Penfield has stated that lesions of the temporal lobe usually give rise to macular-splitting hemianopia, whereas those of the occipital lobe frequently cause hemianopia with macular sparing. Dr. Leinfelder has

pointed out that hemianopia with macular sparing is seen with lesions of parts of the visual system other than the occipital lobe. A review of the literature, with particular reference to lesions of the optic chiasm, the optic tract and the temporal lobe shows that macular sparing occurs not only with lesions of the occipital lobe but also with lesions of the temporal lobe, the optic tract and the optic chiasm.

There is, then, the possibility that some fiber system or tract other than one passing through the posterior part of the corpus callosum might explain macular sparing.

Dr. Penfield suggested that the reason we find a macular splitting in our second case is that we have removed or damaged more of the temporal lobe than in the first case. The specimen in the second case is actually smaller than that in the first; yet that is not conclusive evidence that there is not more damage in the temporal lobe, for we do not know what vascular disturbance accompanied or followed the removal of this portion of the brain. It is conceivable that there are softenings in the posterior part of the temporal lobe and that Dr. Penfield may be right in his assumption that greater damage to this lobe is the explanation for the macular-splitting hemianopia in the second case.

PHAKOMA RETINAE AND ADENOMA SEBACEUM

SANFORD R. GIFFORD, M.D.
CHICAGO

Since the first publication in 1921 by van der Hoeve [1] describing the association of benign circumscribed retinal tumors with tuberous sclerosis of the central nervous system (Bourneville), I have been on the watch among clinical and private patients for this curious condition. The first case corresponding at least in part to this syndrome has recently been seen (1939). For some reason, the condition, a hereditary one, seems to occur or to be observed with especial frequency in Holland, so that van der Hoeve has collected records of a large number of affected families from his own experience. He coined the name "phakoma" to describe congenital tumors arising in various tissues which were distinct from nevi by the absence of nevus cells. It is from the Greek "phakos," the term for "mother spot," or birthmark, and has nothing to do with the root word for lens. He has shown that at least three different but somewhat allied conditions could be logically grouped under this name, with distinguishing subtitles. The tumors are in all cases benign in that they do not metastasize, but they are usually multiple and may have serious or fatal results, according to their location. These three conditions are: angiomatosis (von Hippel and Lindau), affecting the retina and brain, especially the cerebellum; neurofibromatosis (von Recklinghausen), affecting the skin, optic nerves and cranial nerves, and tuberous sclerosis (Bourneville), affecting the skin, retina and brain, especially the cerebrum. Van der Hoeve has shown that retinal tumors occasionally occur in patients with neurofibromatosis [2] which resemble those seen in tuberous sclerosis in some respects, while the presence of greatly enlarged vessels suggests a relation with angiomatosis. (The names suggested by van der Hoeve for the three conditions are phakomatosis von Hippel-Lindau, phakomatosis von Recklinghausen and phakomatosis Bourneville.) He has more recently described a "fourth phakomatosis." [3] This is the so-called Sturge-Weber syndrome, in which patients are

From the Department of Ophthalmology, Northwestern University Medical School.

1. van der Hoeve, J.: Arch. f. Ophth. **105**:880, 1921; **111**:1, 1923; Tr. Ophth. Soc. U. Kingdom **52**:380, 1932.

2. van der Hoeve, J., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 612; footnote 1.

3. van der Hoeve, J.: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **51**: 136, 1936; abstracted, Klin. Monatsbl. f. Augenh. **97**:98, 1936.

affected by naevus flammeus, buphthalmos and enlarged cerebral vessels, which result in atrophy of the cerebrum and cerebellum with paralysis or epilepsy. Characteristic changes are seen in roentgenograms of the skull corresponding to the enlarged vessels. As in all the other phakomatoses, a hereditary basis is present, other members of the family being affected with the complete or the partial syndrome. In the case reported by van der Hoeve buphthalmos was not present, but one eye had been crushed by a birth injury and contained a tumor, which was considered to be an atypical glioma.

Tuberous sclerosis, described by Bourneville in 1880, occurs in families afflicted with epilepsy and mental deficiency. Later observers noted the association of tumors in the retina, skin, kidneys, heart, thyroid, mammae and parts of the skeletal system. The mental symptoms usually develop in early childhood and vary from the slightest defects to complete idiocy, usually with epilepsy. Many patients die before the thirtieth year, but some live to an average age. The changes in the central nervous system include diffuse sclerotic degeneration of portions of the brain and localized nodular tumors composed chiefly of glia and atypical ganglion cells. The tumors do not metastasize but develop independently in various places. The changes in the skin have been called "adenoma sebaceum (Pringle)." They are located typically on either side of the nose and on the cheeks and consist in groups of small, yellowish red, rounded nodules originating in the sebaceous glands. There may also be present on other parts of the body small fibromas, café au lait spots and depigmented areas, such as are seen in Recklinghausen's disease. The retinal tumors, first described by van der Hoeve, are white to gray nodules with a peculiar hob-nailed surface. They are in various parts of the fundus, sometimes on the papilla, and vary greatly in size, and several may be present in the same eye. One or both eyes may be affected, and the visual disturbance depends on the location of the tumors. Sections in a case of van der Hoeve showed the tumors to be composed of fibers deriving from the nerve fiber layer and of undifferentiated retinal elements. Nearly all contained cavities filled with serum and red blood cells. Some of the retinal tumors have remained stationary, while others have grown slowly, and in 1 case of van der Hoeve's a tumor was seen in the process of disintegrating, with the formation of secondary tumors on the surface of the retina, apparently by implantation after transportation through the vitreous. Not all affected persons showed all of the stigmas of the syndrome, some members of an affected family presenting only cerebral symptoms, while others were without the cutaneous lesions. In one of van der Hoeve's families, of 9 children, 5 showed signs of tuberous sclerosis of the brain but no retinal tumors, while a sixth showed retinal tumors but no signs of epilepsy or mental

deficiency. He reported other cases in which some members of affected families showed retinal tumors without cerebral symptoms or epilepsy. In all 3 cases reported by Anker and Kveim,[4] typical adenoma sebaceum and characteristic retinal tumors of one eye only were present. The patients showed mental deficiency, which in 1 amounted to complete idiocy, while 1 other was a high grade moron and the third was only slightly underdeveloped but of an epileptic family. All showed bony changes consisting in periosteal thickening and cystic areas.

The only case which could be found in the American literature is that of Messinger and Clarke.[5] Their patient was a man of 20 with

Fig. 1.— Right eye of patient, showing a retinal tumor above the macula and depigmented areas.

epilepsy and adenoma sebaceum, conditions also present in the father. The change in the fundus consisted in a white, shiny mass which obscured the disk, extending forward 5 diopters. The patient died of pneumonia, and sections showed the ocular growth to be composed of primitive glia cells with secondary calcification. There were also typical lesions of tuberous sclerosis in the brain, rhabdomyoma of the heart and lipofibromas of the kidneys. These authors collected reports of 24 cases

4. Anker, M., and Kveim, A.: Acta ophth. **16**:454, 1938.

5. Messinger, H. C., and Clarke, B. E.: Retinal Tumors in Tuberous Sclerosis: Review of Literature and Report of Case, with Special Attention to Microscopic Structure, Arch. Ophth. **18**:1 (July) 1937.

of retinal tumor associated with tuberous sclerosis from the literature, in 4 of which sections were studied microscopically.

For other cases in the literature, the papers of van der Hoeve and of Anker and Kveim may be consulted.

A report of the single case which I have seen follows:

A man of 37, of Irish descent, presented himself because of poor vision in the left eye, known to be present for the past three years. Vision in the right eye was normal, and the eye showed nothing abnormal in the media or the fundus. Vision in the left eye was 5/200 and was unimproved by correction. The fundus showed an almost white, round elevated mass in the retina up and in from the macula. Its surface was smooth except for a pointed elevation toward the vitreous.

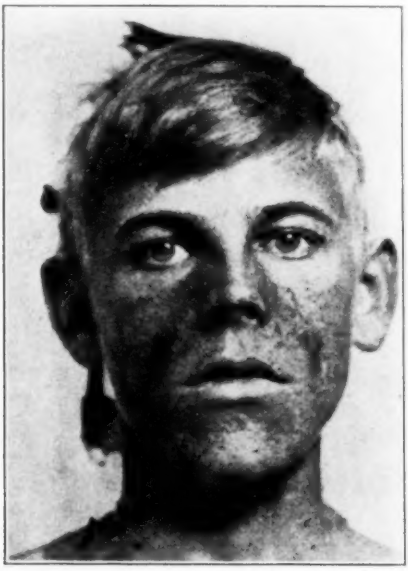

Fig. 2.—Adenoma sebaceum. (A picture of Anker and Kveim's patient, published with their permission.)

It measured ½ papilla diameter and was crossed by several small retinal vessels (fig. 1). It was elevated about 2 diopters above the surrounding retina. There were a few areas of depigmentation with ill defined borders in the macular area and elsewhere in the fundus. Such areas, in association with retinal tumors, were noted in a recent case by Elstad.[6] The skin showed typical adenoma sebaceum, most marked on the cheeks, and also a number of soft pedunculated tumors on the shoulders and arms. Dr. Stillians, head of the department of dermatology, Northwestern University, saw the patient and agreed that the cutaneous condition of the face was adenoma sebaceum while the tumors elsewhere were neurofibromas. Some dermatologists consider that adenoma sebaceum is a variety of neurofibromatosis. Unfortunately, no photographs of the face were

6. Elstad: Acta ophth. **17**:344, 1939.

made, and the patient cannot be located for further examination. The condition resembled almost exactly that shown in Anker and Kveim's case 2, and hence their illustration is used with their permission (fig. 2). The patient showed no signs of mental defect, nor was there a history of mental defect or epilepsy in the family. One sister is known to have poor vision since childhood, but I was unable to get her to come in for examination. No roentgenograms of the bones were made in this case.

From the picture of the fundus and the cutaneous condition, there seems to be no doubt that the condition described by van der Hoeve was being dealt with, although in the present case the mentality was normal. Since the tumors in this condition are benign, there is no indication for enucleation of the affected eye. It is impossible to say how long the retinal tumor was present. In spite of the patient's opinion that poor vision had been present for only three years, it seems likely that the tumor was present from an early age, as this has been true in most of the reported cases.

Although this is the second case to be reported in the American literature, it seems likely that careful examinations of the fundi of families with epilepsy and mental deficiency would show a certain number of similar cases.

CORNEAL OPACITIES IN THE ALASKAN ESKIMO

A POSSIBLE CAUSATION

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In the summer of 1938 I spent several months in Northwestern Alaska as a medical officer on a coast guard cutter, and as a part of my duties I saw about 800 natives with various medical complaints. The natives of the region are Eskimos and persons with a mixture of white and of Eskimo blood. The total population of the villages visited was 3,553 (which included about 100 white persons). The territory covered by the cruise included the coast line from a point 90 miles east of Nome, north as far as Barrow and the islands adjacent to the mainland.

A condition frequently found was corneal opacity, causing much individual disability. The condition was found in 57 patients, was bilateral in all, was of about equal extent in both eyes and involved large portions of the cornea. The opacities were of varying degree of density. There was no patient who gave a history of trauma. Opacities were encountered in all age groups. The opacities were striate and always involved the lower segments of the cornea to the greatest extent and became more nebulous superiorly.

Tuberculosis, which was frequently seen, failed to explain the malady. Syphilis is so rare as to be almost unknown in the Eskimo race. Deficiency of vitamin A could produce corneal opacity, but no other manifestation of vitamin deficiency was noted in my studies of 800 natives for general medical conditions. Furthermore, the natives eat large amounts of walrus and seal meat, which contain adequate quantities of vitamin A to protect against serious deficiency disease of this type. There may be a relation between the severe winters and the opacities, but I saw several patients with inflamed eyes and early opacities several months after the last of the cold weather.

After studying several cases in an effort to establish the causation, I had the good fortune to have a patient with epiblepharon come to me complaining of her eyes. She was a healthy 19 year old Eskimo with typical mongolian features, including a broad nose, a round head and face, wide interpupillary distance, epicanthic folds, high cheek bones and hair which was straight, coarse and black. On examination of the eyes there was bilateral epiblepharon of such extent that the margins of both lower lids were obscured. When the patient looked downward the lashes

of the lower lids were forced backward, brushing the corneas. This occurred in the central portion only when the patient looked downward a matter of 10 degrees, but when she dropped her line of vision more than 10 degrees all the cilia were applied to the globe. Nebulous opacities were present over the lower central portions of both corneas. There were small striate areas in the lower portions of both corneas which stained with fluorescein. Mild pericorneal injection was present. The anterior chambers were clear. Visual acuity was about normal in both eyes. Otherwise, examination of the eyes gave negative results.

Epiblepharon is a disease not mentioned in most textbooks on ophthalmology and even in some medical dictionaries. As defined by McCool in Berens' textbook,[1] it is a "congenital anomaly characterized by a horizontal fold of the lower eyelid which covers the eyelid margin and part of the eyelashes when the patient looks down. When the condition is marked a permanent inward turning of the cilia may be produced."

Epiblepharon was first described by von Ammon[2] in 1841 in a study of infants and young children. It has since been recorded by others, all having studied the condition in infants and in children, in whom the condition is temporary. Herrenschwand[3] in 1917 reported the first case in an adult that I am able to find in the literature. The case reported by Pillat[4] is well studied. It was that of an adult mongolian who had a well defined epiblepharon, which resulted in some roughening of the lower portions of the corneas but without opacity.

To return to my case of epiblepharon: Since I expected to see the patient at a later date, I adopted the following line of treatment. All of the lashes of the lower left lid were extracted. The right eye was kept as a control. When the patient was seen a week later the left eye had recovered a healthy appearance. There was no staining of the cornea. The condition in the right eye was unchanged, and there were still the small staining areas of the cornea.

On this second visit the lashes were extracted from the right lower lid, and the following procedure was carried out on the left eye. A fold of skin was removed from the left lower lid corresponding to the fold of the epiblepharon and horizontal to the margin of the lower lid. The

1. McCool, J. S.: Diseases of the Eye Lids, in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 349.

2. von Ammon, F. A.: Der Epicanthus und das Epiblepharon; zwei Bildungsfehler der menschlichen Gesichtshaut, J. Kinderkr. **34**:313, 1860.

3. von Herrenschwand, F.: Ueber Entropium congenitum und Epiblepharon, Klin. Monatsbl. f. Augenh. **58**:385, 1917.

4. Pillat, A.: Marked Epiblepharon with Retroflexion of the Eyelashes of Both Lower Lids in an Adult Mongolian, Arch. Ophth. **1**:315 (March) 1929.

edges of the wound were approximated in such a way as to obliterate the epiblepharon and tacked to the deep tissues of the lid. When the patient was seen a month later there were no subjective or objective findings. The operation done had accomplished its purpose, and the same procedure was carried out on the right lid.

At about the same time this case of epiblepharon was encountered, another was seen, again in an adult. In this case no opacity was seen on either side. A few scattered striate areas of staining were encountered in both corneas. The physical findings were about the same as in the first case. I used the described plastic procedure on this patient on my first visit. When the patient was seen again a month later the eye operated on had regained a normal appearance but the other eye was unchanged. On this visit the right eye was operated on, but I am not able to report a result as I did not call at the village again.

Two additional cases of epiblepharon of such extent that the lashes brushed the corneas were found, but circumstances did not permit much study or corrective procedures of a permanent nature.

Unfortunately my observations came late in my cruise. Before that time no special attention was paid to the type of facies or to the anatomic structure of the lids beyond the usual examination of the external eye. Naturally all this was changed after studying my first case. In all the later cases the facies was typically mongolian, and in all of them well developed epiblepharon was present, though often the disease was not of such extent that lashes were turned backward.

It may be argued that such superficial trauma is unlikely to produce corneal opacity, since opacity does not result unless Bowman's membrane is traumatized. However, repeated insults to the corneal surface over a long period could produce opacity of the cornea such as has been described.

From my observations I think there is little doubt of a relation between the corneal opacities of the Eskimos and the epiblepharon with which many of them are afflicted. However, further study will be required to demonstrate this relation scientifically.

CONCLUSIONS

1. Bilateral opacities of the cornea, covering extensive portions, are relatively common in the Alaskan Eskimoes.

2. Epiblepharon, a fold of skin horizontal to the margin of the lower lid, is common in the Alaskan Eskimoes. In some cases the condition is sufficiently developed to turn the cilia of the lower lid backward against the globe.

3. Further study will be required to demonstrate scientifically a relation between these two factors.

LOCALIZATION OF INTRAOCULAR FOREIGN BODIES

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TRENTON, N. J.

Localization of intraocular foreign bodies is of prime importance not only for their successful extraction but for the preservation of the greatest amount of vision. Without knowing the exact location of a foreign body, one would be at a loss as to where to make an incision and in which direction to insert the point of the magnet or, if one is dealing with a nonmagnetic foreign body, where to search for it. In fact, a roentgenogram taken without a localizer is of little value because it cannot show even whether the foreign body is within the eyeball.

In the past there has been excellent work done by localizers such as the one devised by the late Dr. Sweet. However, one of the drawbacks to their use has been their cost, which has prevented many a hospital and roentgenologist from owning one, especially considering the fact that it is not frequently needed. The other method of localizing a foreign body, by a special roentgen technic, requires five roentgenograms and depends too much on the good behavior of the patient. I have attempted to devise an instrument which is simple in operation and low in cost. The device is a frame made to fit over the eyeball, as shown in figures 3 to 6. The smaller circle measures 12 mm. (inside diameter) and fits around the cornea. The inside diameter of the larger circle is 22 mm. and the outside diameter 24 mm. The latter circle fits over the sclera, on which it reaches to a point 5 mm. in front of the equator. The distance between the planes of the two circles is 6 mm. The four cross bars divide the eyeball into four quadrants. Since the larger circle covers the whole circumference of the eyeball, any foreign body shown within this circle in the anteroposterior view is bound to be in the eyeball, provided the plate of the lateral view also shows it to be so. Bearing in mind that the inside diameter of the eyeball is 22 mm. and knowing the distance between the corneal and the scleral circle of the instrument, one can readily calculate the depth of the foreign body shown in the lateral plate.

TECHNIC FOR THE USE OF THE LOCALIZER

A 4 per cent solution of cocaine hydrochloride is instilled in the injured eye three times, a few minutes apart. The localizer is placed

Read before the Section on Ophthalmology at the Ninety-First Annual Session of the American Medical Association, New York, June 13, 1940.

The experimental part of this technic was done at the Mercer Hospital, with the cooperation of Dr. Irvin Davenport, the radiologist of the hospital.

Fig. 1.—Roentgenogram taken without a localizer. This is of little value because it does not show whether or not the foreign body is within the eyeball.

Fig. 2.—Roentgenogram taken without a localizer (lateral view). Localization is impossible.

Fig. 3.—The localizer shows the foreign body 2 mm. below the horizontal line and 5 mm. toward the temporal side from the center of the cornea.

Fig. 4.—Lateral view of foreign body shown in figure 3. The foreign body is seen about 5 mm. from the limbus.

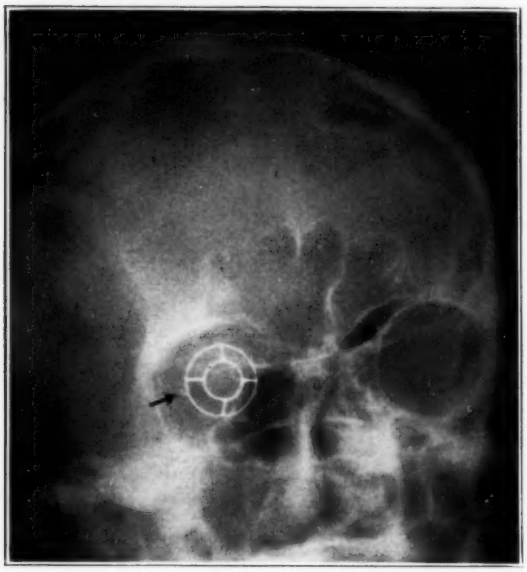

Fig. 5.—The localizer shows the foreign body lying outside the eyeball and 5 mm. below the horizontal line.

Fig. 6.—In this lateral view the foreign body is seen about 10 mm. from the limbus, measured from the vertical diameter of the corneal circle, on account of the picture not having been taken in exactly the lateral position.

over the eyeball with the cross pieces at 90 and 180 degrees. The lids are closed over the instrument and held together with a piece of adhesive plaster. Two roentgenograms are taken, one in exactly the anteroposterior and the other in exactly the lateral position. The pictures are taken with the patient in the sitting position, with the good eye open and fixating straight at a target. In the case of uncontrollable children the lying down position will have to be resorted to and both eyes closed with adhesive plaster. The plates are immediately developed, and if the pictures are satisfactory the pieces of adhesive plaster and the instrument are removed from the patient's eyes. If a radiopaque foreign body has entered the eyeball, it will be localized by study of the two plates.

The possibility of a minute body falling behind the wires of the instrument and thus being obscured in the picture is a remote one, because if such a thing should happen when the anteroposterior plate is made most probably it could not happen when the lateral plate is made and vice versa. However, to eliminate any possibility whatsoever, especially when more than one foreign body is suspected and only one is found in the plate, a flat picture without the localizer should be taken; then if a second body is seen, the localizer should be reintroduced, this time with the cross bars at slightly different angles, and another set of pictures taken. My belief is that this will never be necessary, especially in civil practice.

BILATERAL SUBCONJUNCTIVAL LYMPHOID INFILTRATION

REPORT OF A CASE

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Noninflammatory subconjunctival tumors of the episclera are not common. They are usually lymphomas or leukemic nodules. The following is a report of such a case:

About ten months ago Mr. J. McC., white, aged 59, was told by his wife that his right eye was red and swollen. He never had any ocular pains or

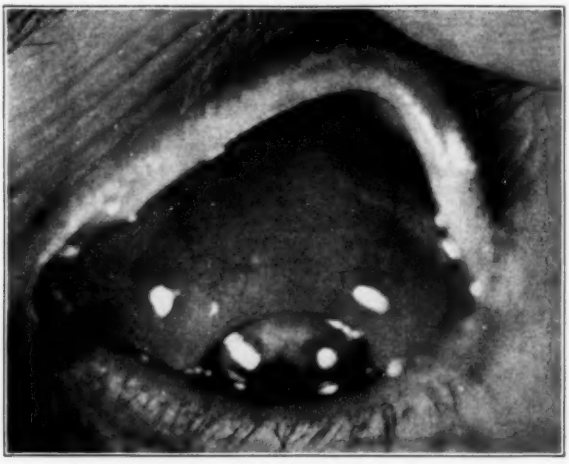

Fig. 1.—Appearance of the patient's left eye.

pasting of the lids. About three weeks later the same condition was noticed in the left eye. He was observed at the clinic and hospital of the Jersey City Medical Center in the service of Dr. F. X. Brophy. On his admission the bulbar conjunctivas of both eyes were raised by an extensive salmon-colored or reddish thickening and showed dilated tortuous conjunctival vessels (fig. 1). This pericorneal condition was present in the upper halves of both globes extending as far back on the sclera as could be seen. The mass was sharply demarcated and the conjunctivas and scleras at the margin of the tumors appeared normal. Around the cornea in each eye the tumor overhung the limbus, but without evidence of corneal involvement. The conjunctivas of the upper and lower lids as well as in the retrotarsal folds were normal. On palpation the subconjunctival mass was

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solid and of a hepatic consistency. There was no limitation of motion in any field or diplopia. The lacrimal glands were apparently normal. Transillumination gave negative results. The ocular tension, the fundi and the visual fields were normal.

Examination showed chronic sinusitis, severe dental caries and slightly enlarged right submental and bilateral axillary and inguinal nodes. A roentgenogram of the skull and sinuses showed disease of the antrums. The dental picture showed

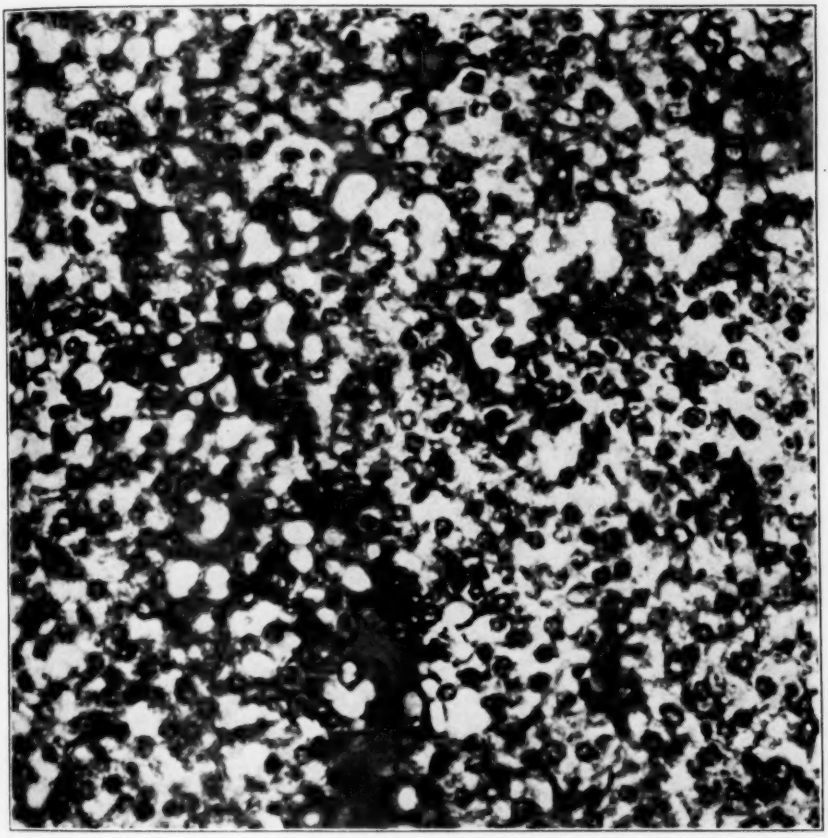

Fig. 2.—Section of the ocular mass.

extensive caries with apical pathologic involvement and retained roots. Roentgenograms of the chest and long bones were negative.

A Mantoux test with a 1:1,000 dilution of tuberculin gave a faintly positive reaction. The results of vaccination with Bacillus melitensis and of a control test were negative after forty-eight hours. Laboratory tests showed basal metabolic rates of + 33 and + 58 per cent. There was moderate secondary anemia, a hemoglobin content of 76 per cent (Sahli) and ascending leukocytosis, the leukocyte count ranging from 11,100 to 30,100 cells per cubic millimeter. Of note in the differential count were 12 to 14 per cent band cells and 1 to 2 per cent

juvenile myelocytes. Chemical examination of the blood, including determinations of the phosphorus, calcium and phosphatase, gave negative results. The provocative Wassermann test was negative. The blood serum agglutination for tularemia and undulant fever were negative. Urinalyses gave negative results. Bence Jones protein was absent. Smears from the tumor mass were negative for leprosy. A biopsy specimen of the bone marrow from the sternum, by aspiration, was normal.

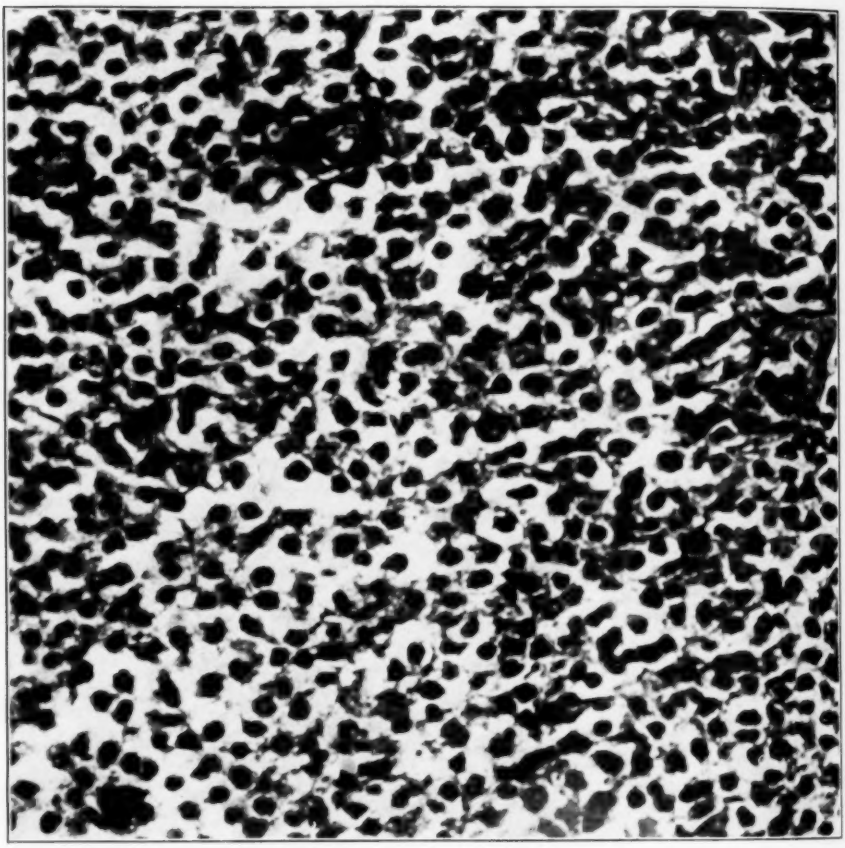

Fig. 3.—Section of the submental lymph node.

During the patient's hospitalization the temperature, pulse rate and respiratory rate were normal. Locally, the treatment consisted of the use of antiseptic drops and hot boric acid compresses. Systemically, a series of injections of foreign protein, phenyl salicylate, azosulfamide (neoprontosil; disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonate), 33 per cent mercury inunctions and saturated solution of potassium iodide were given consecutively.

Surgically, a biopsy specimen from the left eye was obtained, the mass being easily separated from the normal sclera below. A biopsy specimen from the right

submental lymph node was secured. Section of the ocular mass showed myelocytes, a few plasma cells and numerous undifferentiated blast cells with occasional reticulum (?) (fig. 2). In the less cellular portion of the tumor were numerous blood spaces and collagen fibers. The amyloid test was negative. A section of the submental lymph node showed atypical lymphatic leukemia (fig. 3). (Both biopsy specimens were studied by Dr. F. W. Stewart, of New York.)

The patient went home during the Christmas holidays in good health. On Dec. 30, 1939 he entered the hospital comatose and died of acute fulminating pneumococcic meningitis, type VIII. Death occurred within thirty hours of the onset of his illness.

A diagnosis of leukemia without invasion of blood and bone marrow (?) and lymphoma (?) was made.

PERIPHERAL VASCULAR PICTURE IN RETINITIS PIGMENTOSA

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DETROIT

The etiology of retinitis pigmentosa has engaged the interest of ophthalmologists almost from the day Helmholtz' ophthalmoscope became available in 1852. The entity was first described by Donders, of Utrecht, and the first anatomic investigation was done by Maes in 1861. Landolt [1] followed with a report in 1872, and soon there arose two schools of thought, divided on the fundamental question of whether the neuroepithelial degeneration was primary or secondary to choroidal sclerosis.

As time advanced, other factors have been brought forward as etiologic. These include glandular deficiencies, trauma of light, avitaminosis and toxicity. Of these, the theory of toxicity alone has withstood critical analysis. The strength of this theory has been considerably enhanced by experimental work showing that various substances injected subcutaneously at some distance from the eye, and also in the eye, will act selectively on retinal epithelium and produce pigmentary degeneration.

Sugita's [2] work on the effect of bile on visual purple is significant, as it showed that bile will dissolve visual purple. When visual purple is exhausted, rods die. In his pathologic sections Sugita found cholesteatose deposited in the retinal pigment cell layer and partial degeneration of this layer.

Takahashi [3] found faulty hepatic function in his 12 cases. This work is inconclusive, because after finding 12 patients with retinitis pigmentosa in a large series of patients with disease of the liver he concluded that

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From the Division of Ophthalmology, Henry Ford Hospital.

1. Landolt, E.: Anatomische Untersuchungen uber typische Retinitis pigmentosa, Arch. f. Ophth. (pt. 1) **18**:325-348, 1872.

2. Sugita, Y.: Experimentelle Untersuchungen über die Wirkung der Galle und ihrer Bestandteile auf das Auge, speziell auf den Lichtsinn und den Sehpurpur, nebst Bemerkungen über meine Sehpurpur-Lösungsmethode, Arch. f. Ophth. **116**: 653-666, 1926.

3. Takahashi, T.: Ueber die Funktionsstörung der Leber bei Pigmentdegeneration der Netzhaut, Arch. f. Ophth. **116**:143-152, 1925.

all retinitis pigmentosa is accompanied by hepatic disease. The fault of this reasoning is apparent.

Nevertheless, these investigations give considerable impetus to the toxin or exogenous theory of causation, and we feel that our own work in no way contradicts it; in fact, it offers considerable in the way of support.

This then leaves the following three theories to be considered: 1. There is an abiotrophic status present in the rods which causes an early death. 2. The death of the rods is due to choroidal sclerosis. 3. There is no sclerosis, but the rods die from some exogenous influence.

Collins [4] in 1922 strongly advocated the abiotrophic concept and popularized the use of Gowers' term of nerve tissue death as one to be applied to death of the retinal neuroepithelium. Later this conception was supported by Keeler,[5] who showed that in certain blind white mice the retinal neuroepithelium was absent or rudimentary and that the condition was transmitted as a mendelian recessive characteristic. Additional support is gained by Leber's [6] analysis of the literature prior to 1915, in which he stated that the microscopic studies of Gowin, Stock, Ginsberg and Suzanna ruled out the possibility of choroidal sclerosis as a causative factor.

Verhoeff [7] described a case of retinitis pigmentosa in 1931 and expressed the belief that the choroid was not responsible for the changes. He found endarteritis of the choriocapillaris but ascribed it to senile changes, as his patient was 64 years of age. He expressed the opinion that there was an exogenous influence of some sort that was not well understood.

It is apparent that much is yet to be discovered concerning the etiology of this disease. It also seems most likely that there is either some destructive agent transmitted to the choriocapillaris and thence across Bruch's membrane to the detriment of the rods, or that the choriocapillaris is unable to nourish the neuroepithelium through some abnormality in itself which is not a sclerosis (and therefore not evident in pathologic specimens) but which nevertheless produces an anoxia great enough to cause death.

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4. Collins, E. T.: Hereditary Ocular Degenerations; Ophthalmic Abiotrophies, in International Congress of Ophthalmology, Washington, 1922, Philadelphia, William F. Fell Company, 1922, vol. 1, pp. 103-143.

5. Keeler, C. E.: The Inheritance of a Retinal Abnormality in White Mice, Proc. Nat. Acad. Sc. **10**:329-333, 1924.

6. Leber, T., in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1915, vol. 7, p. 1076.

7. Verhoeff, F. H.: Microscopic Observations in a Case of Retinitis Pigmentosa, Arch. Ophth. **5**:392-407 (March) 1931.

With this thought in mind, an evaluation of the general vascular picture in 9 cases of retinitis pigmentosa was undertaken. The study consisted of slit lamp evaluation of the capillaries at the limbus, a microscopic study of the capillaries in the nail bed and occlusion index curves. The studies of the capillaries at the limbus were made by us and the studies of nail beds and the occlusion index by the staff of the division of general medicine.

The amount of spasticity, tortuosity, segmentation and decrease in the size of the vessels was designated as from 1 to 4 plus, 1 plus denoting definite deviation from the normal and 4 plus denoting the extreme of abnormality as seen in capillary examination in any disease of the blood vessels.

Two charts were made. The first shows the abnormal occlusion indexes of the first 5 cases. The second shows the curves of cases 6, 7, 8 and 9.

The table summarizes the findings. Note that there was a marked abnormality of the nail bed and limbus in the last 4 cases, in which the occlusion indexes were normal.

A brief report on the examinations made in these cases is offered:

CASE 1.—An unmistakably pathologic curve was obtained. The vessels at the limbus were narrow, sparse and very spastic. In the nail bed the subsized capillaries were decreased in number. They were nubby in type, tortuous and exhibited grade 3 spasm. Segmentation was also noted, but the flow was moderately rapid. The feet were warm and of normal color. Pulsation was felt in the right dorsalis pedis artery, but the posterior tibial artery was scarcely palpable.

CASE 2.—There was some inequality of caliber in the vessels, some right angle turning and tortuosity. In the nail bed the capillaries were of approximately normal caliber, but there was occasional dilatation of the venous sides of the loops with the formation of capillary nests. The skin was cool, and good pulsation was present in all the peripheral arteries. Note the rigidity of the curve. A diagnosis of polycythaemia vera was also made in this case.

CASE 3.—The vessels of the limbus were slightly tortuous and spastic. In the nail bed a normal number was seen, but definite tortuosity was present with dilatation of most of the loops. The flow was sluggish. There was no segmentation. The left dorsalis pedis artery was not definitely occluded, but the pulse was small. The posterior tibial artery was not definitely felt. The occlusion index in this case was pathologic in that cooling did not occur rapidly enough and the rise after heat stimulus was neither high nor rapid enough. The curve is the least pathologic of the abnormal group, but the delayed cooling time and the sluggish response to heat justify its inclusion.

CASE 4.—The curve obtained in this case is quite flexible. The vessels at the limbus showed only a little tortuosity. In the nail bed there was a normal number but with increased tortuosity and spasm of the terminal loops, the flow being quite sluggish; larger vessels were palpable.

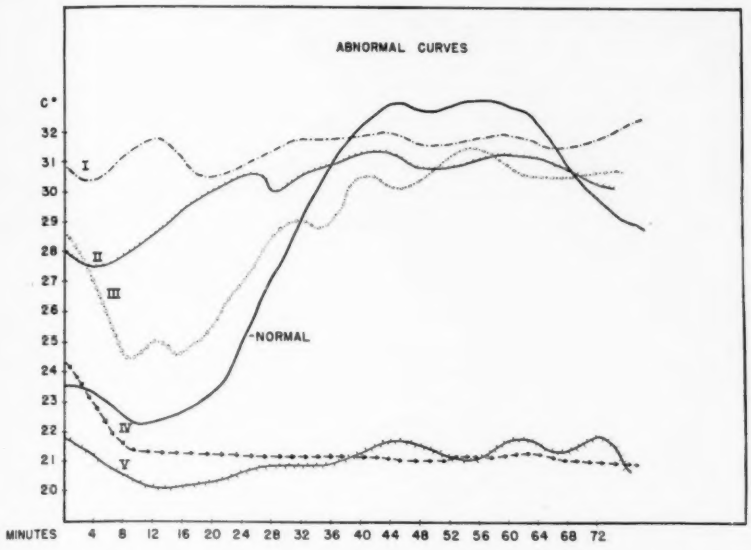

Chart 1.—Curves showing abnormal occlusion indexes in cases of retinitis pigmentosa.

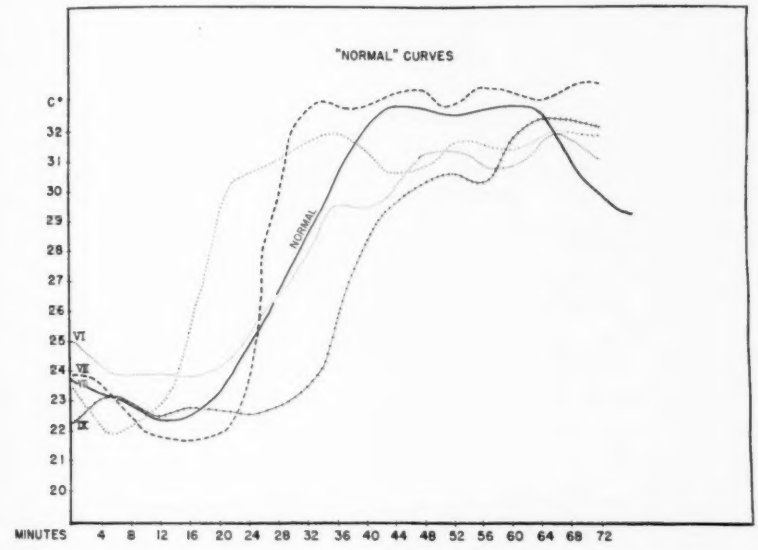

Chart 2.—Curves showing normal occlusion indexes in cases of retinitis pigmentosa.

CASE 5.—The vessels at the limbus showed spasm, right angle branching and tortuosity. In the nail bed there was a decrease in the number of capillaries with slightly increased tortuosity and some dilatation of the terminal loops. The flow was slow, but no definite spasm was noted. Both feet were cold, but pulsation in the peripheral vessels was good. The occlusion index curve was very abnormal.

CASE 6.—The vessels at the limbus showed only moderate tortuosity and right angling but were spastic at some places. There was a reduction of capillaries of the nail bed and most were arranged in the direction of the nail. There were a few figures of eight, and a number of the capillaries showed more or less segmentation with spasm. A 2 plus occlusion of the right dorsalis pedis artery was present, but the occlusion index was normal.

CASE 7.—There were spasm and tortuosity of the vessels at the limbus. Some of the dilatations caused by the spasm even approached the caliber of a saccular aneurysm. In the nail bed there was marked tortuosity of the capillaries, and no normal loops were seen. Some showed considerable dilatation and others marked segmentation and spasm. The skin and pulsation were normal.

Summary of the Data in Nine Cases of Retinitis Pigmentosa

Case	Age	Blood Pressure	Condition of Blood Vessels at Limbus	Condition of Blood Vessels in Nail Bed	Occlusion Index
1	26	130/78	+++	+++	++++
2	51	176/100	++	+	+++
3	32	135/85	++	++	+
4	21	125/80	++	++	+++
5	30	128/76	+	+	++++
6	30	128/72	++	+++	0
7	22	115/80	+++	+++	0
8	19	125/80	++++	++++	0
9	28	134/84	+++	+++	0

CASE 8.—The patient, a French girl of 19, had a decrease of vision to perception of light because of cataracts. She presented at the limbus a marked picture of segmentation and spasm, although her curve was normal. In the nail bed the capillaries were greatly reduced in number. Those seen were only short pieces lying parallel instead of perpendicular to the nail. In some places they were rosette shaped with dilatation. A moderate amount of spasm was present. The temperature of both feet was normal. The patient gave a typical history of blanching of the fourth and fifth fingers on exposure to cold. The typical three color phase of Raynaud's disease was described, but an attempt to bring about the effect with hot and cold water was unsuccessful.

CASE 9.—The vessels of the limbus showed marked tortuosity and some spasm. In the nail bed there was a normal number of capillaries, which were very tortuous. There were spasm and slowed flow but no dilatation. Pulsation in larger vessels was normal.

A summary of these findings is presented in the table. The most marked changes in the vessel as shown by direct examination were found in the last 4 cases, but the occlusion index was normal. I have no explanation for this discrepancy.

SUMMARY AND CONCLUSION

In 9 cases of retinitis pigmentosa the peripheral vascular picture at the limbus and the nail bed was studied by biomicroscopy and the occlusion index was determined.

In all 9 cases there was unmistakable evidence of a generalized peripheral vascular disturbance of the capillary type.

There is enough alteration of metabolism at the chorioretinal threshold due to this capillary abnormality to produce anoxia and subsequent death of the neuroepithelium.

ANTICATARACTOGENIC ACTION OF CERTAIN NITROGENOUS FACTORS

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A report from this laboratory published in the ARCHIVES in 1938[1] reviewed the progress to date on the use of galactose as a means of producing experimental cataract in rats. Studies were reported which indicated that rations high in protein were protective and that those low in protein hastened the lenticular changes due to galactose. Yudkin and Geer[2] have recently verified the effect of the low protein but questioned the action of the high protein ration. Verification of our previous findings with high protein rations has been followed by further research directed toward the identification of the protective agent. The quest has led into several blind alleys, but finally into one which leads deeper into the nitrogen story. It would appear that some factor in protein or derived from it is responsible for this inhibitory effect even in the presence of the injurious agent, galactose. The present paper reports the various nitrogenous factors tested and the results which lead us to believe that the protective agent is nitrogenous and that it may be some specific amino acid or group of amino acids.

EFFECT OF THE AMOUNT OF PROTEIN IN THE RATION

The experimental procedures and rations were similar to those previously reported. The three levels of protein (casein) were fed in the following rations (values given in per cent):

Ingredient	Low Protein	Medium Protein	High Protein
Casein	5	15	45
Galactose	25	25	25
Starch U. S. P. (corn starch)	50	40	10
Hydrogenated oil	9	9	9
Cod liver oil	2	2	2
Salt mixture (Osborne and Mendel's)	4	4	4
Brewers' yeast	5	5	5

From the Department of Home Economics Research, Massachusetts State College.

This paper is contribution no. 370 of the Massachusetts Agricultural Experiment Station.

In this series the growth of rats on the "low" protein ration was distinctly retarded, and they naturally ate less food than litter mates on adequate protein rations. Nevertheless cataract developed in them more rapidly in spite of a reduced intake of galactose. The rats on the "high" protein diet grew at approximately the same rate as those on the "medium" protein diet. A direct comparison of litter mates showing the effect of different levels of protein on incidence and rate of development of cataract is shown in chart 1. The incidence of mature cataract was reduced to less than half by the increase from 15 to 45 per cent protein, and the time required was more than doubled.

An understanding of the charts will be facilitated by knowing a few more details of the way in which the experiments have been conducted

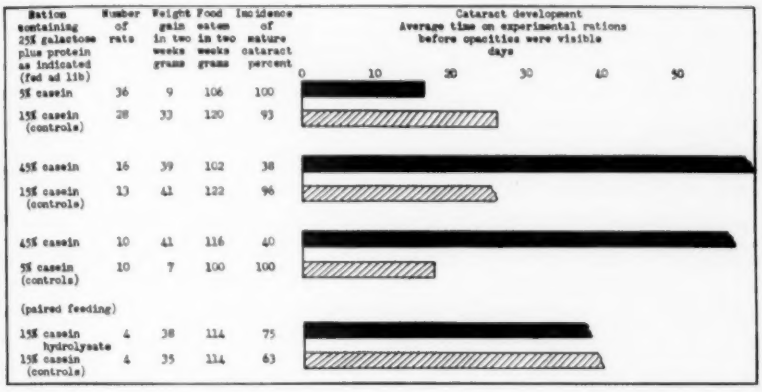
Ration containing 25% galactose plus protein as indicated (fed ad lib)
Number of rats
Weight gain in two weeks grams
Food eaten in two weeks grams
Incidence of mature cataract percent
Cataract development
Average time on experimental rations before opacities were visible
days
0
10
20
30
40
50
5% casein 36 9 106 100
15% casein (controls) 28 33 120 93
45% casein 16 39 102 38
15% casein (controls) 13 41 122 96
45% casein 10 41 116 40
5% casein (controls) 10 7 100 100
(paired feeding)
15% casein hydrolysate 4 38 114 75
15% casein (controls) 4 35 114 63

Chart 1.—Influence of casein at different levels and of casein hydrolysate on the development of galactose cataract.

and interpreted. All rats were started on experiments at 25 or 26 days of age, and only litter mates are compared in any of the experiments reported in this paper. Galactose was fed at the 25 per cent level in all rations, the only variation being in the nitrogenous constituents with reciprocal alteration in the level of the corn starch. Ad libitum feeding with accurate records of food intake were replaced in later experiments with a paired feeding technic. The 15 per cent protein ration given in the tabulation was the skeleton ration modified as indicated in the various other experiments.

1. Mitchell, H. S., and Cook, G. M.: Galactose Cataract in Rats, Arch. Ophth. **19**:22 (Jan.) 1938.

2. Yudkin, A. M., and Geer, H. A.: An Investigation of Experimental Cataracts in the Albino Rat: Clinical Implications, Arch. Ophth. **23**:28 (Jan.) 1940.

Feeding tests on each litter were terminated at the end of nine or ten weeks, whether or not cataract had developed. Earlier experience had demonstrated that relatively few changes occurred in the eyes after the first ten weeks, and the expensive nature of these experimental rations made longer observations unwarranted. Figures for growth and food intake are given for two weeks only, because that was the maximum period for which data were available for all of the animals. These figures are of interest chiefly when a reduced food intake may be responsible for a slower development of cataract or when an aggravation of the injury was apparent on low protein rations in spite of a reduced food intake.

The bars in the charts indicate the average number of days the rats were on the experimental ration or the number of days before mature cataract developed. Cataract failed to develop in one or more rats in each of several groups during the experimental period. In such cases the number of days they were on the experimental ration was the figure used in deriving the averages, although mature cataract had not occurred in that time. Obviously a correct average would be somewhat greater than the figure calculated in this way. Bars with jagged ends indicate such averages. Bars with square ends are used wherever 100 per cent incidence of mature cataract in the group made it possible to calculate the average time exactly. When there were not the same number of rats from each litter on each ration being compared, the averages were weighted to give equal weight to each litter represented. This correction was necessary for fair comparison because of the litter variation in susceptibility to cataract. Thus, the average time for the development of cataract in two different groups of rats on the same ration may vary because different litters are represented in the averages. So also when food intakes are controlled, the development of cataract may be delayed in proportion to the reduction in the amount of galactose consumed, but litter mate controls are reduced to the same extent.

EFFECT OF VITAMIN SUPPLEMENTS

The possibility that commercial casein or other sources of protein might carry adsorbed vitamins which would be significant in these experiments has been investigated from various angles. When vitamin-free casein was used in place of commercial casein in the ration, cataract developed in the same length of time, but certainly no earlier, as might have been expected had the vitamins in commercial casein been significant. Furthermore, egg albumin coagulated, washed and dried for feeding tended to delay the development of cataract more than the casein; yet this dried egg albumin is a source of protein notably free from vitamins with the exception of riboflavin, which has been further investigated.

The feeding of massive doses of either thiamine chloride (vitamin B_1) or riboflavin (vitamin G) afforded no protection against galactose injury, as reported by us [3] in 1938 and verified by Yudkin and Geer in 1940.[2] Earlier work had shown that a known deficiency of these two as well as other vitamin factors repeatedly failed to hasten or aggravate the development of cataract due to galactose. There is no reason to believe that the lenticular injury due to high blood sugar (galactose) is in any way related to the deficiency of a known vitamin factor.

EFFECT OF HYDROLYSIS

The possibility that some protein structure itself rather than the constituent amino acids might be significant was approached by feeding a casein enzymic hydrolysate furnished by Meade Johnson & Co. The paired feeding of two litters on cataractogenic rations containing the casein and casein hydrolysate at equivalent nitrogen levels gave evidence of equal protection against cataract (chart 1). The possibility of certain significant impurities being carried on the protein molecules would also seem to have been eliminated by this procedure. Thus it appeared that some derivative of protein hydrolysis or metabolism should be considered.

EFFECT OF SULFHYDRYL AMINO ACIDS

Earlier work from this laboratory [4] and similar findings reported by Bellows [5] gave encouragement to the idea that cystine might exert a specific protective action against injury to the lens by galactose. These observations fitted well with the earlier reports that there was a decrease in the sulfur content of cataractous lenses.[6] Later attempts to repeat these findings with more carefully controlled experiments have been disappointing. When 2 per cent of cystine or methionine were added respectively to the 15 per cent protein ration, the protection, if any, was not commensurate with that afforded by added protein providing a similar amount of these amino acids. With later experiments using a controlled food intake, there appeared to be some slight delay in development of cataract with cystine but almost none with methionine (chart 2).

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3. Mitchell, H. S., and Cook, G. M.: Effect of Riboflavin and Thiamin Chloride upon the Cataractogenic Action of Galactose, Proc. Soc. Exper. Biol. & Med. **39**:325, 1938.

4. Mitchell, H. S., and Cook, G. M.: Influence of Protein or Cystine Intake on Cataract-Producing Action of Galactose, Proc. Soc. Exper. Biol. & Med. **36**: 806, 1937.

5. Bellows, J. G.: Biochemistry of the Lens: IX. Influence of Vitamin C and Sulfhydryls on Production of Galactose Cataract, Arch. Ophth. **16**:762 (Nov.) 1936.

6. O'Brien, C. S., and Salit, P. W.: Chemistry of the Lens, Am. J. Ophth. **16**:863, 1933.

A failure of other laboratories as well as our own to control the food intake of rats on cataract-producing rations may have led to wrong interpretations. When rats refuse to eat, as they invariably do when higher levels of certain amino acids or other distasteful supplements are incorporated in the ration, they fail to ingest enough galactose at any one time to cause the characteristic changes in the lens. The apparent protection which is observed is due merely to partial starvation. Neither Yudkin and Geer [2] nor Bellows [5] has indicated that controlled or paired feeding technic was used, which prompts one to question the significance of the results reported. Results in this laboratory have been much more satisfactory since these precautions have been taken.

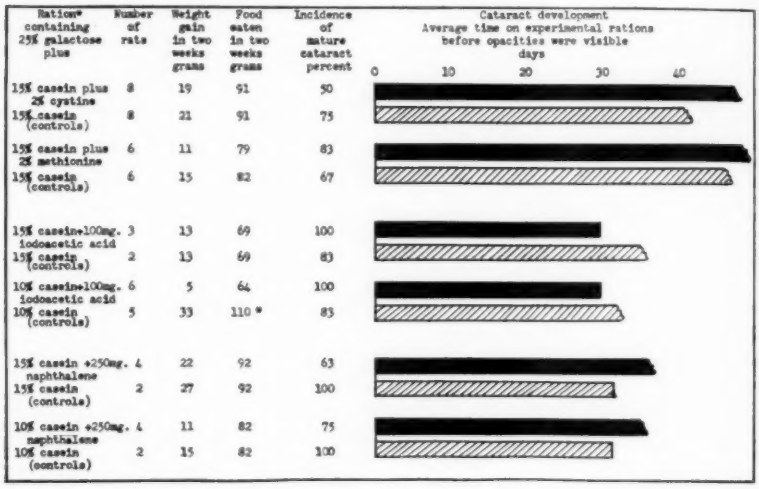

Chart 2.—Influence of the sulfhydryl amino acids on the development of galactose cataract. The asterisk indicates that paired feeding was employed except for a few rats in the group so marked.

The use of chemically pure iodoacetic acid (100 mg.) in a 10 per cent casein ration has been reported by White [7] to make unavailable the major portion of the sulfhydryl amino acids and to result in retarded growth. This device was used as a means of studying the influence of cystine and methionine on the cataractogenic action of galactose. Growth was retarded in rats receiving the iodoacetic acid with 10 per cent of casein but not with 15 per cent. Cataract development was 100 per cent in both groups, and the time was a little shorter on the average than in the control animals which received no iodoacetic acid. The differences are suggestive rather than highly significant but are in the direction which would indicate that the sulfhydryl amino acids might have some part to play (chart 2).

7. White, A.: Iodoacetic Acid and Sulfur Metabolism, Science **86**:588, 1937.

The detoxication of naphthalene is supposed to require sulfur and thus to deprive the animal body of a portion of its sulfur supply, according to Stekol.[8] Moreover, naphthalene has been reported by Bourne[9] to cause cataract in rabbits. If sulfur compounds were significant in this pathologic change and if there were a connection between these two findings, naphthalene might be expected to aggravate galactose cataract. Growth was only slightly retarded by the addition of 250 mg. of chemically pure naphthalene in 100 Gm. of either the 10 or 15 per cent casein ration, but cataract was delayed, if anything, rather than hastened (chart 2). Paired feedings throughout this experiment support the validity of these findings in spite of the limited number of animals used.

Thus from three lines of attack it must be concluded that the sulfhydryl amino acids are of questionable significance in the control or inhibition of the cataractogenic action of galactose in rats.

EFFECT OF SIMPLER NITROGENOUS COMPOUNDS

Urea, the end product of nitrogen metabolism, has been shown to be an available source of nitrogen in certain species of animals and not in others. The question was raised as to whether such a simple form of nitrogen could possibly play a significant role in replacing protein in these cataract-producing rations. Urea (chemically pure) at 1, 5 and 10 per cent levels was incorporated in a 5 per cent casein ration containing the usual 25 per cent of galactose. Records of rats on these three levels of urea indicated little, if any, utilization of urea as a source of nitrogen for growth. Growth was about equally retarded in all groups, including the controls which received the 5 per cent casein ration without added urea. On the other hand, there is some evidence that the higher levels of urea afforded slight protection against cataract (chart 3). This is evident if one studies the findings and takes notice of the fact that although the litters used with the 10 per cent urea trial happened to be the most susceptible, even so the time for the development of cataract was longer than in the other groups. Obviously it is impossible at this point to theorize as to just how the urea or its nitrogen exerts this action. The possible synthesis of certain amino acids from urea and glycogenic end products is frequently postulated in biochemical discussions.

Choline, because of its demonstrated influence on lipoid metabolism, was suggested as of possible significance here. When fed at levels of

8. Stekol, J. A.: Studies on Mercapturic Acid Synthesis in Animals: Effect of Naphthalene on Growth of Rats as Related to Diets of Varying Sulfur Content, J. Biol. Chem. **121**:87, 1937.

9. Bourne, M. C.: Effect of Diet on the Nature of Ocular Lesions Produced by Naphthalene, Brit. J. Ophth. **17**:210, 1933.

1, 2, 4 and 8 per cent, incorporated in a 5 per cent casein cataract-producing ration, choline exerted no protective action whatever. The reduced food intake of rats on the 8 per cent choline level precluded the continuance of controlled feeding with this one group. This partial starvation was in itself responsible for the observed delay in the development of cataract. The results obtained with the three lower levels of choline are shown in chart 3. The retardation of development of cataract in both experimental and control groups with increasing amounts of choline was due to the decreased food intake resulting from distaste for the choline.

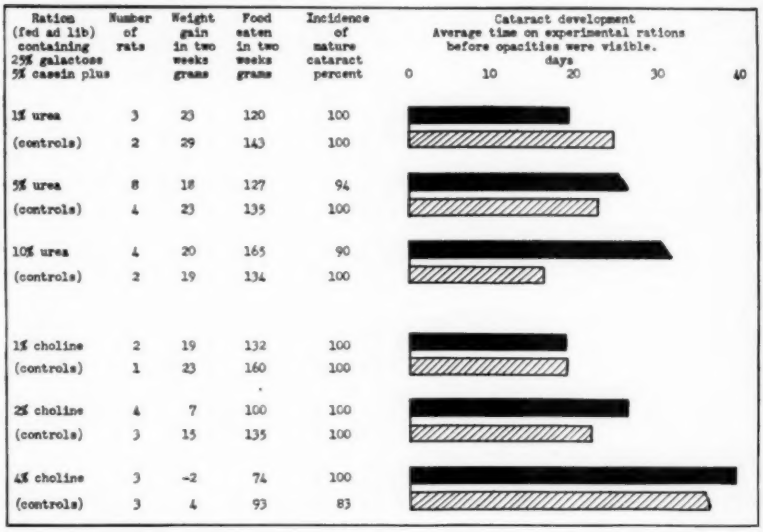
Ration (fed ad lib) containing 25% galactose 5% casein plus
Number of rats
Weight gain in two weeks grams
Food eaten in two weeks grams
Incidence of mature cataract percent
Cataract development
Average time on experimental rations before opacities were visible.
days
0
10
20
30
40
1% urea 3 23 120 100
(controls) 2 29 143 100
5% urea 8 18 127 94
(controls) 4 23 135 100
10% urea 4 20 165 90
(controls) 2 19 134 100
1% choline 2 19 132 100
(controls) 1 23 160 100
2% choline 4 7 100 100
(controls) 3 15 135 100
4% choline 3 -2 74 100
(controls) 3 4 93 83

Chart 3.—Influence of replacement of part of the protein with urea or choline on the development of galactose cataract.

EFFECT OF DEAMINIZATION OF PROTEIN

The treatment of proteins with certain acids is known to destroy or change certain amino acid groups and therefore make the protein less adequate for growth. Deaminized casein prepared by the treatment of commercial casein with nitrous acids according to the method outlined by Dunn and Lewis [10] is incapable of supporting growth of rats when fed as the sole source of nitrogen in the diet. When it was fed as two thirds of the 15 per cent protein in a ration of which one third was commercial casein, growth was extremely slow and approximately the same as for the rats on the 5 per cent protein (casein) ration. If this loss of

10. Dunn, M. S., and Lewis, H. B.: The Action of Nitrous Acid on Casein. J. Biol. Chem. **49**:327, 1921.

the amino acid complex necessary for growth should also lessen the protective action of the protein in respect to cataract, it would furnish a clue as to the nature of the protective factor.

Contrary to expectation, the ration containing two thirds of its protein from the deaminized casein was more protective than either the 5 or the 15 per cent commercial casein protein controls. The usual explanation, that the loss or destruction of certain amino acids accounts for the failure to support growth, offers no hypothesis concerning the present finding. Whatever amino acids were destroyed by treatment with nitrous acid, they were certainly not the protective agents. What further alteration may have taken place in the protein molecule to account for the increased protection has not yet been determined. Further work is being conducted along this line and will be reported in detail elsewhere.

Egg albumin similarly treated with nitrous acid was also unable to support growth but showed marked protection against the galactose injury in the few animals in which it was tried, under carefully controlled conditions.

EFFECT OF HEATED PROTEIN

The prolonged exposure of certain proteins to dry heat has long been known to alter their nutritive value. Casein, wheat gluten and egg albumin have each been heated for ninety-six hours at 125 C. and subsequently incorporated into cataractogenic ration as the sole source of protein. The inhibition of growth due to the heat treatment was apparent in all three, but in the casein alone was there a marked loss of protection against cataractous changes. This work is still in progress and will be reported in detail elsewhere.

COMMENT

The further study of galactose cataract in rats and the results of feeding the various nitrogenous factors reviewed in this report lead to some interesting hypotheses which are being investigated. The fact that a protein hydrolysate functions in a similar capacity to the whole protein molecule suggests that some amino acid or group of amino acids may be responsible. The sulfhydryls do not appear to be the group in question, and the simpler nitrogenous substances tried, urea and choline, do not offer much of a lead for further work. The supposed loss or alteration in structure of certain amino acids when protein is treated with nitrous acid could not explain the increase in protective action observed with deaminized casein unless some new compound is formed. On the other hand, the dry heat seems to destroy the protective action of casein. Since two modified forms of casein, both of which retard growth, have

been shown to have opposite effects in the cataract work, it would appear that the factors necessary for growth promotion and for cataract inhibition are not the same.

Although the type of cataract produced in young rats by feeding galactose may be different from senile or other types of cataract in human beings, there seems to be one question in common: What nutritional factors, if any, will maintain the lens and its capsular membrane in a state least susceptible to injury or degeneration? Perhaps the capsular membrane is particularly susceptible to damage in these young rats. Even so, if the protective dietary factor is effective in the presence of the injurious agent in susceptible animals, it would seem that the health or resistance of the lens or capsular membrane had been improved. If the blood sugar had been markedly lowered by the feeding of protein, this fact alone could have accounted for less lenticular injury. Since this was not the case, it may be assumed that the protective action does, in some way, concern the lens or its capsular membrane. If some factor in protein is thus protective against galactose injury, is it not possible that it might exert a similar function against other injurious agents and in other mammalian species?

SUMMARY

The incidence and rate of the development of galactose cataract in rats were increased by a protein deficiency and decreased by a high protein ration as previously reported.

An enzymic casein hydrolysate exerted the same protective action as commercial casein.

Neither of the sulfhydryl amino acids cystine and methionine seemed to offer specific protection against galactose cataract when observed with a paired feeding technic.

The simpler nitrogenous compounds urea and choline were not effective substitutes for protein in its anticataractogenic capacity.

Deaminized casein was more protective and heated casein less protective than commercial casein.

The significance and possible application of these findings are discussed.

Clinical Notes

SOME OBSERVATIONS ON CROSSED CYLINDERS

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The superiority of the Jackson crossed cylinder technic in determining the exact axis of astigmatism has been adequately stressed and demonstrated photographically, mathematically and practically. Not much is said about its advantages in determining the exact amount of cylinder required in any given case. I believe this is due to the fact that as one makes the series of indicated changes in the cylindric lens in the trial frame, one must be continually altering the amount of the spherical component of the combination in order to keep the general visual acuity up to a sufficiently high level for fine differences to be perceptible.

If the accommodation has been paralyzed in a case of compound hyperopic astigmatism, the visual acuity will be increased by the addition of plus spheres up to a figure that corresponds to the spherical error plus one half of the cylindric error, provided proper test objects are used. The same idea may be expressed by formula by assuming that the total error would be corrected by a glass such as a + A sph. ⌒ + B cyl., axis C; if one starts adding plus spheres to such an eye, the vision will improve until one has placed a + (A + ½B) sph. before the eye. This is due to the fact that if a + A sph. alone is used, the anterior linear focus is on the retina; if a + (A + B) sph. is used, the posterior linear focus is on the retina, but if a + (A + ½B) sph. is used, the portion of the conoid of Sturm just anterior to its center and where a cross section of the conoid is a circle (circle of least confusion) is on the retina.

If, with this spherical lens in place, one starts adding plus cylinders, the posterior linear focus is drawn forward but the position of the anterior linear focus is not altered, because when congeneric lenses are used the location of the anterior linear focus is determined by the spherical lens alone and the position of the posterior linear focus is determined by the spherical and cylindric lenses combined. When by the use of the plus cylinder the posterior linear focus is drawn forward a certain amount, the circle of least confusion is also moved forward off of the retina one half of that amount. (This is not exactly true if computed by linear measure, but it is true if measured in diopters.) At this stage better vision would be obtained if the spherical lens in the trial frame were reduced by an amount equal to one-half the numerical value of the plus cylinder used.

If one should choose to use minus cylinders, one would push the anterior linear focus back and the circle of least confusion back one-half as much and would not alter the location of the posterior linear focus. In such a case better vision would result from adding a plus sphere of one-half the numerical strength of the minus cylinder used, thus keeping the circle of least confusion on the retina.

All of this means that after finding the spherical lens that gives best vision, when a cylinder is used it should be combined with a contrageneric sphere of one-half the dioptric value of the cylinder. This is exactly what is offered by the crossed cylinders of Jackson, so that if a series of such crossed cylinders are mounted so they can be placed in the trial frames, the number of lens changes necessary can be reduced by one-half, the circle of least confusion can always be kept on the retina and all of the confusing results that come from the removal of the circle of least confusion forward or backward from the retina can be avoided. In this way the anterior linear focus is pushed back exactly the same number of diopters as the posterior linear focus is pulled forward, each focus all the time getting shorter and closer together until at last they become coincident points on the retina in the plane formerly occupied by the circle of least confusion. All that remains is to reduce this combination of sphere and crossed cylinders to the simplest spherocylindric combination. This process is too simple to require comment.

The equipment required is not as extensive as might be thought. I use the usual crossed cylinder with the handle at 45 degrees to the axes as the indicator for determining whether the cylindric correction should be augmented or diminished. I find that four crossed cylinders mounted to go in the trial frames are adequate for almost any case, the strengths being + and —0.12, 0.25, 0.37 and 0.50 cyl. and the total cylindric strength being, respectively, 0.25, 0.50, 0.75 and 1.00 cyl.

810 Medical Arts Building.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

OCULAR CONDITIONS ASSOCIATED WITH CLINICAL RIBOFLAVIN DEFICIENCY

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AND
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CLEVELAND

ARIBOFLAVINOSIS AND CATARACT

In 1931 the occurrence of cataract as a result of a deficiency of riboflavin in the diet of young rats was described.[1] Many significant papers have followed this observation without making close correlation between the type of change observed in the rat eyes and the changes seen with the slit lamp in any type of human cataract. It should be emphasized, however, that structural differences between the human and the rat lens not observed with the slit lamp may exist. It is not permissible to deprive infant human beings of riboflavin so completely as is necessary to produce cataracts in young rats. It is equally improbable that one will ever see patients deficient in riboflavin alone to the extent necessary to produce cataracts in young rats. Fundamental as these studies have been, no specific changes in the human lens have yet been described as "ariboflavinosis opacities." The incidence of cataract among persons whose diet is obviously almost void of riboflavin does not warrant the inference that a cause and effect relation to the deficiency exists. Until more conclusive evidence is presented, it can but be emphasized that a well balanced diet is essential.

INTERSTITIAL KERATITIS

Recently a report has been published concerning the use of riboflavin for the treatment of 2 patients with ocular manifestations diagnosed as

* Fellow in biochemistry and recipient of the George Angell and the S. M. A. stipends for research in ophthalmology.

From the Western Reserve University School of Medicine and the University Hospitals of Cleveland, Department of Surgery, Ophthalmologic Service (Dr. Johnson).

1. Day, P. L.; Langston, W. C., and O'Brien, C. S.: Cataract and Other Ocular Changes in Vitamin G Deficiency, Am. J. Ophth. **14**:1005, 1931.

syphilitic interstitial keratitis.[2] Since the slit lamp appearance described differs considerably from the vascular pattern (resembling a large unleafed elm branch, without conspicuous anastomosis or Rouget cells) which one of us (L. V. J.) was familiar with in Boston and now observes in Cleveland, an extended quotation from this article follows:

> A white girl, aged 9, with a diagnosis of congenital syphilis, had been under continuous antisyphilitic treatment for 15 months. . . . The slit lamp showed that the entire cornea was covered with fine interlacing and anastomosing vessels located just beneath the epithelium. There was extensive interstitial invasion and the posterior membrane was also covered with a fine vascular plexus. . . . All antisyphilitic treatment was stopped and riboflavin (5 mg. daily) was given. Nine days later it was evident that the diffuse interstitial opacity was less and that many of the smaller vessels were empty. Three weeks thereafter photophobia was absent and the opacities had cleared to such a degree that the child was reading without her glasses. . . .

A portion of the report of the other case follows:

> A colored female, aged 24, with a diagnosis of acquired syphilis, had been previously treated with antisyphilitics at various times, but for 5 months the treatment had been continuous and intensive. . . . Examination with the slit lamp showed a picture comparable in most respects to that described for case 11 [the preceding case] . . . Antisyphilitic treatment was stopped and riboflavin (5 mg. daily) was given. . . . Two and a half months later no patent vessels could be found and only a few small areas of interstitial exudate remained.

From these cases the following conclusions were drawn:

> (1) Syphilis may have no causal relationship to keratitis; their association may be a pure coincidence.
>
> (2) Syphilis may be a contributing or precipitating cause of keratitis . . . if the bodily state with reference to riboflavin is mildly but imperceptibly impaired from a cause such as ariboflavinosis, corneal involvement might be expected with syphilis.
>
> (3) Syphilis may be a primary cause of keratitis.

During the fall of 1938 it was our opinion that we might experimentally demonstrate what the foregoing authors call the second "possibility," using rabbits infected with syphilis and placed on a diet deficient in riboflavin.[3] Unsuccessful attempts were discontinued when in a girl 9 years of age, who had been given 6 mg. of riboflavin daily for over three months, a more severe interstitial keratitis developed in the second eye during treatment than had been present in the first. At the University Hospitals of Cleveland patients with interstitial keratitis are usually given riboflavin, or more recently elixir of vitamin B complex, but the results are not spectacular.

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2. Kruse, H. D.; Sydenstricker, V. P.; Sebrell, W. H., and Cleckley, H. M.: Ocular Manifestations of Ariboflavinosis, Pub. Health Rep. **55**:157, 1940.

3. Johnson, L. V., and Eckardt, R. E.: Is the Onset of Interstitial Keratitis Related to Riboflavin Deficiency? Arch. Ophth. **23**:631 (March) 1940.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.

Explanation of Figures

Fig. 1.—Acute solitary marginal infiltrate with minimal limbal vascularization. These lesions disappear entirely within fourty-eight hours after intravenous injection of riboflavin and within seventy-two hours after oral administration of riboflavin. Adequate diet usually prevents recurrence.

Fig. 2.—Multiple marginal infiltrates on a site previously involved. Note that the remnants of the vessels persisting from the previous vascularization cause the new infiltrates to occur beyond their free tips, rather than near the limbus.

Fig. 3.—High lights show facets remaining after the absorption of the infiltrates following administration of riboflavin. It cannot be seen that much of the vascularization has disappeared.

Fig. 4.—Progress of a severe, untreated rosacea ulcer, so called. Note that this infiltrate has developed central to the vessels remaining from a previous attack.

Fig. 5.—The infiltrate is larger and has progressed toward the center of the cornea. When viewed with the slit lamp, it is observed that none of the vessels quite touch the infiltrate; if the progress of the infiltrate is followed, it is seen that it clears as the vessels grow toward its edge. An acute chalazion has developed. We have observed Demodex in the contents of these chalazia. They must be opened during the acute phase if the mite is to be recovered.

Fig. 6.—Very severe rosacea keratitis, so called.

It is apparent, however, that interstitial keratitis with "fine interlacing and anastomosing vessels located just beneath the epithelium" and with "the posterior membrane covered with a fine vascular plexus" represents a type of corneal disease with which we are not familiar, and a dietary deficiency may coexist. For this type of pathologic process, the improvement following the use of riboflavin is reported as very promising.

ROSACEA KERATITIS (SO CALLED)

In December 1939 we submitted for publication data which we had prepared during the previous two years concerning the possible cause and effect relation between riboflavin and so-called rosacea keratitis.[4] Further observations on those patients and on 14 additional patients have not altered the conclusions that it contained. It is now our policy to administer about 9 mg. of riboflavin intravenously during the first week, divided as daily or triweekly injections, and thereafter to continue with elixir of vitamin B complex, so that the daily amount of riboflavin is about 3 mg. Simultaneously the diet is adjusted to provide more than customary amounts of milk and liver, and as the remission continues the amount of supplementary elixir of vitamin B complex is diminished. Patients are advised to resume the administration of the vitamin B complex if early symptoms recur. The practice of applying chemical cauterizing agents to the cornea has been discontinued at the University Hospitals of Cleveland. An excellent description of the pathologic involvement of the limbic blood vessels has been given by Doggart.[5] Recently Sydenstricker and others[6] described in detail changes in the cornea which appear to be identical with rosacea keratitis. Their term, "dietary keratitis," appears to initiate a new designation for the condition familiar to ophthalmologists as rosacea keratitis, so called.

PATHOLOGIC CONDITIONS OF THE CORNEA ASSOCIATED WITH PELLAGRA

Unfortunately, we have not had the opportunity to observe the cornea of pellagrins sufficiently to allow an opinion concerning the ocular pathologic changes. The following quotation from Sebrell[6a] is exceedingly interesting.

---

4. Johnson, L. V., and Eckardt, R. E.: Rosacea Keratitis and Conditions with Vascularized Cornea Treated with Riboflavin, Arch. Ophth. **23**:899 (May) 1940.

5. Doggart, J. H.: Ocular Rosacea, Tr. Ophth. Soc. U. Kingdom **50**:98, 1930.

6. Sydenstricker, V. P.; Sebrell, W. H.; Cleckley, H. M., and Kruse, H. D.: The Ocular Manifestations of Ariboflavinosis, J. A. M. A. **114**:2437 (June 22) 1940.

6a. Sebrell, W. H.: Nutritional Diseases in the United States, J. A. M. A. **115**:851-854 (Sept. 7) 1940.

The very early writers on pellagra, such as Soler in 1791 and Rampoldi in 1885, refer to such eye symptoms as inflammation of the cornea, corneal ulcers and opacities, and among the early writers on pellagra in this country, Clark in 1909 reported pain in the eyes, conjunctivitis, failing vision and iritis, and Whaley saw photophobia, mydriasis and superficial inflammation of the cornea among other eye lesions in pellagrins. It seems not unlikely now that, in view of the close association frequently seen between riboflavin deficiency and pellagra, many of these lesions may have been due to riboflavin deficiency. Last year Spies, Vilter and Ashe again called attention to such lesions and stated that more than 70 per cent of the patients in their nutrition clinic with frequent recurrences of pellagra, beriberi or riboflavin deficiency also have visual disturbances.

These ocular lesions were described briefly by Spies, Vilter and Ashe.[6b]

A lesion of the eyes characterized by bulbar conjunctivitis, lacrimation, burning of the eyes and failing vision has responded in some instances after the same treatment [riboflavin]. It is too early to interpret these observations, since in other cases similar lesions respond to large doses of carotene.

It then appears that so-called rosacea keratitis represents a naturally occurring metabolic disorder in which the utilization of riboflavin is somehow impaired to a variable extent, and that mild or subclinical disease is produced in pellagra patients who probably would be able to utilize riboflavin normally if it were supplied in the diet.

MULTIPLE OBSTRUCTION OF MEIBOMIAN GLANDS

Of indirect interest is the observation that we have been able to demonstrate demodecidae (face mites) in the expressed contents of acute multiple obstruction of meibomian glands, the material being placed in a drop of cedar wood oil or glycerin and viewed immediately with a low power microscope. We have not been successful in demonstrating these organisms in chronic operable chalazia. The observations by György [7] of the association between rat pediculosis and ariboflavinosis is to be recalled. Clinical evaluation of such a condition treated with riboflavin is difficult, but it is our impression that there is definite value.

TWILIGHT BLINDNESS

Pock-Steen [8] has observed twilight blindness associated with leiodystonia and sprue, which improved following administration of ribo-

6b. Spies, T. D.; Vilter, R. W., and Ashe, W. F.: Pellagra, Beriberi and Riboflavin Deficiency in Human Beings: Diagnosis and Treatment, J. A. M. A. **113**:931-937 (Sept. 2) 1939.

7. György, P.: Pediculosis in Rats Kept on Riboflavin-Deficient Diet, Proc. Soc. Exper. Biol. & Med. **38**:383, 1938.

8. Pock-Steen, P. H.: Eye Symptoms in Patients with Leiodystoma and Sprue: Twilight-Blindness, Geneesk. tijdschr. v. Nederl.-Indië **79**:1986, 1939.

flavin. With this condition, we have had no experience. We have observed no marked changes in the curve obtained with the biophotometer before and after riboflavin was given to several of our patients with rosacea keratitis.

SUPPORTIVE TREATMENT FOR CORNEAL ULCERS

In a preceding issue of the ARCHIVES OF OPHTHALMOLOGY,[4] we discussed the importance of riboflavin in the metabolism of avascular tissues such as the cornea, in which Warburg's yellow enzyme is effective in transferring hydrogen from the tissue cells to the capillaries at the limbus. To be certain that insufficient oxidative enzymes are not delaying healing, we are using vitamin B complex as supportive treatment for all types of corneal ulcers. It might be emphasized here that factors additional to riboflavin are concerned with the healthy state of the cornea. For example, György [9] observed that the deficiency of pantothenic acid in the diets of rats caused a high incidence of corneal ulcers, which tended to progress to perforation and endophthalmitis. Addition of pantothenic acid to the diet is followed by a definite tendency to healing. The inclusion of pantothenic acid in elixir of vitamin B complex makes the use of this preparation of definite theoretic advantage as a general supportive measure for corneal ulcers.

9. György, P.: Biochem. J., September 1940, to be published.

CORRECTIONS

In the article by Drs. F. H. Verhoeff and G. Victor Simpson entitled "Tubercle Within Central Retinal Vein: Hemorrhagic Glaucoma; Periphlebitis Retinalis in Other Eye," in the October issue (ARCH. OPHTH. **24:** 645, 1940), the first line under the center head "Report of a Case" should be inserted after the fifteenth line, which reads "No hemorrhages were seen in any part of the retina."

In the article by Dr. F. H. Verhoeff entitled "Phi Phenomenon and Anomalous Projection," which appeared in the August issue (ARCH. OPHTH. **24:** 247, 1940), the third word from the end in the seventh line on page 250 should read "right" instead of "left."

News and Notes

EDITED BY W. L. BENEDICT

GENERAL

Ophthalmologica.—The September number of *Ophthalmologica* is the beginning of the hundredth volume of this journal. It began as the *Zeitschrift für Augenheilkunde*, under the editorship of Kuhnt and von Michel. It was then continued by Meller and Behr until the present series began and the name was changed to the present one.

It is now being edited by Brückner (Basel, Switzerland) and Weve (Utrecht, Netherlands) and is emphasizing international cooperation.

All good wishes and congratulations are extended to the present editors.

PERSONAL

Appointment to Dartmouth Eye Institute.—Dr. Walter B. Lancaster, of Boston, has been appointed chief of staff of the Dartmouth Eye Institute and will assume his new duties on November 1. Dr. Lancaster has practiced his specialty in Boston since 1897. In addition to his practice as consulting ophthalmic surgeon, he is at present an associate in ophthalmology at the Harvard University Medical School and consulting ophthalmic surgeon at the New England Hospital for Women and Children. He will relinquish this work to devote himself fully to his new post at Dartmouth.

Dr. Lancaster has long specialized in the field of physiologic optics and was instrumental in founding at Dartmouth the department of research in physiologic optics, from which the present Dartmouth Eye Institute has grown. The college honored him in 1939 with the Doctorate of Science.

Dr. Lancaster was graduated from Harvard University in 1884 and from the Harvard Medical School in 1889. He was ophthalmic surgeon successively at the Boston City Hospital and the Massachusetts Eye and Ear Infirmary for three decades and since 1928 has continued his work in a consulting capacity. He was formerly instructor in ophthalmology at the Harvard Medical School and has held his present associateship there since 1933.

Dr. Lancaster is past president of the American Ophthalmological Society, the American Board of Ophthalmology, the American Academy of Ophthalmology and Otolaryngology, and the New England Ophthalmological Society. As chairman of the ophthalmologic section of the National Conference on Nomenclature of Disease, he devised the standard naming of diseases of the eye, and it was he who gave the name "aniseikonia" to the disease of unequal vision, for which the Dartmouth Eye Institute has discovered methods of diagnosis and treatment. During the World War he was head of the department of ophthalmology at the aeronautical research laboratories at Mitchell Field, New York, a background of experience which is expected to be of great value in the

research program which the Dartmouth Eye Institute is now conducting at the Pensacola naval air base for several branches of the government air service.

Dr. Lancaster is a member of the executive committee of the Advisory Board of Medical Specialties, a fellow of the American College of Surgeons and a member of the American Medical Association, the Optical Society of America, the National Research Council, the American Academy for the Advancement of Science, the Illuminating Engineering Society, the Massachusetts Medical Society and the four ophthalmological societies of which he is past president.

Dr. Edwin B. Dunphy has been appointed clinical professor of ophthalmology at Harvard Medical School. He succeeds Dr. John H. Waite.

RECENT PERIODICALS

Boletin del Hospital oftalmológico de Ntra. Sra. de la Luz.—This is a new ophthalmologic journal, published in Spanish in Mexico, D. F. Following the movement in other institutions, the hospital will publish in a special journal the papers and interesting case reports of the members of the staff.

The first number gives the history of the institution, which is the first special hospital for diseases of the eye opened in Mexico since 1876. From that time to the present its size and importance have greatly increased. The bulletin contains in its first number an interesting article by Dr. R. Silva on filariosis (onchocercosis) of the eye, another by Dr. Torres Estrada on the surgical treatment of pterygium and two others on exenteration bulbi and opacities of the pupil.

Besides the *Boletin*, there is in Mexico the old ophthalmologic journal *Anales de la Sociedad mexicana do oftalmología y oto-rino-laringología*, which is now in its fifteenth volume and fourth series.

That two special journals can live and have enough material for publication speaks highly of the activity and progress of Mexican ophthalmology.

Ophthalmologia ibero Americana.—With this name has appeared in Brazil a new ophthalmologic journal, directed by Prof. M. E. Alvaro, of São Paulo. As its name implies, it covers all Latin-American countries and is printed both in Spanish and in Portuguese. Its purpose is the diffusion of ophthalmology in the countries of Iberic languages, bringing to them the latest advances in the specialty. The journal is published every three months by a cooperative society of physicians and contains, beside original articles, a copious section of abstracts from journals of all countries, reviews of societies, book reviews and news and notes.

The journal has about 80 pages and is well printed and illustrated. The list of collaborators includes prominent ophthalmologists of Latin America, the United States and Europe.

Our best wishes and every success to the new journal in its task of approximation of the Spanish-speaking and Portuguese-speaking ophthalmologists in this hemisphere.

Obituaries

F. PARK LEWIS, M.D.
1855-1940

F. Park Lewis died on September 10, after a brief illness, in his eighty-fifth year. He was born in Hamilton, Ontario, Canada, May 19, 1855, and graduated from the Pulte Medical College in 1876. He then took up postgraduate work in New York, London, Paris and Vienna, and settled in Buffalo as an ophthalmologist.

From the first Dr. Lewis became interested in the general problems of preservation of sight and the care of the blind, and his greatest accomplishments were along these lines.

In 1902 Dr. Lewis was appointed by the governor of New York as chairman of a state commission to study the causes of blindness. He was appointed chairman of a second commission in 1905, with an appropriation of $10,000 to make a survey of the same nature and to study means of preventing blindness. The formation of a New York State Committee for the Prevention of Blindness in 1908, which later grew into the present national society, was the outgrowth of this pioneer work.

Dr. Lewis was chiefly responsible for the founding of the National Society for the Prevention of Blindness in 1908 and the International Association for Prevention of Blindness in 1929. The American society had its headquarters in New York, and the International Association maintains a secretariat in Paris. He served as vice president of both organizations since their founding and took an active part in the direction of their policies as a member of the executive committees until his death.

The Leslie Dana Gold Medal, which is awarded annually by the St. Louis Society for the Blind in recognition of outstanding achievements in the prevention of blindness and the conservation of vision, was presented to Dr. Park Lewis in 1928; and a special gold medal was presented to him by the International Association for Prevention of Blindness at its last annual meeting in London, in April 1939, on the tenth anniversary of this association which he helped to found. Buffalo recognized him as one of her outstanding citizens in 1933, when he was awarded the chancellor's medal, an honor awarded annually to the citizen considered most deserving of community recognition.

He was president of the New York School for the Blind at Batavia, N. Y., for the past forty years and was one of the editors of the *American Journal of Ophthalmology*. He was a member of the American Ophthalmological Society and of many other medical societies and contributed important papers at the meetings, where he was a regular attendant.

Surviving are his wife, Mrs. Grace Moseley Lewis, and three daughters, Mrs. Lyman A. Bliss, Miss Dorothea Park Lewis and Miss Frances Park Lewis.

ARNOLD KNAPP.

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ARNOLD KNAPP.

Correspondence

A SERRATED DISCISSION KNIFE

To the Editor:—In the August issue of the ARCHIVES (page 372) Dr. F. C. Parker describes a serrated discission knife, made for him by E. B. Meyrowitz Company. For over twenty years I have employed a serrated Ziegler knife, which, however, is quite different from, and I am sure much more efficient than, the knife depicted by Dr. Parker. In my knife, as shown in the accompanying illustration, the serrations are curved, somewhat resembling those of a bread knife, and immeasurably finer than those in Dr. Parker's knife. These serrations were made by me purposely, and unfortunately I soon forgot how I made them. Fearing some disaster to this knife, I have attempted to duplicate it, but I have never been able to produce a knife nearly as efficient. Several years ago I presented the problem to E. B. Meyrowitz Company,

Finely serrated Ziegler discission knife; × 11.8.

and about two years ago to the Storz Company, but neither of these instrument makers solved it. Evidently the E. B. Meyrowitz Company failed to inform Dr. Parker of their attempt. The Storz Company made for me a knife apparently almost identical with that depicted by Dr. Parker, but I found it useless for my purposes and returned it to the makers.

For his knife, Dr. Parker stated: "This discission needle is not a remedy for all the ills associated with the needling operation, but I feel that it has some advantages over a smooth blade in many instances." For my knife I can make a much stronger statement, namely, that experience with it for over twenty years has proved it to be far superior to a smooth, sharp Ziegler knife for cutting not only the densest but the most delicate membranes. I hope that in the near future some one will find a way to improve on, or at least duplicate, my knife.

F. H. VERHOEFF, M.D., Boston.

Questions and Answers

PROBLEM IN REFRACTION

To the Editor:—A brown-eyed girl of 14 came to me a year ago complaining of headaches. She was wearing:

R: —2.00 sph. ⌒ —0.37 cyl., 90 axis
L: +0.12 sph. ⌒ +0.62 cyl., 90 axis

She had esophoria of 3 prism diopters with the Maddox rod. With homatropine and cocaine cycloplegia the retinoscopic reading was found to be:

R: +0.25 sph. ⌒ —1.00 cyl., 10 axis
L: +0.50 sph. ⌒ —0.62 cyl., 175 axis

On subjective examination, she chose:

R: —1.50 sph. ⌒ —0.75 cyl., 10 axis
L: +0.12 sph. ⌒ —0.50 cyl., 175 axis

Atropine was given, and a new retinoscopic reading was found of:

R: —0.12 sph. ⌒ +0.50 cyl., 90 axis
L: +0.50 sph. ⌒ +0.87 cyl., 90 axis

With this correction she stated she could see almost nothing. She was given a prescription of:

R: —1.25 sph. ⌒ +0.50 cyl., 100 axis
L: —0.25 sph. ⌒ +0.75 cyl., 85 axis

She stated this time that her glasses were fine but that she had to take them off for distance. After the instillation of atropine, a retinoscopic reading was made of:

R: —0.67 sph. ⌒ +1.00 cyl., 100 axis
L: +0.37 sph. ⌒ +0.25 cyl., 85 axis

The patient chose subjectively:

R: —2.00 sph. ⌒ +0.75 cyl., 90 axis
L: +0.12 sph. ⌒ +0.37 cyl., 85 axis

With this she had vision of 20/20 in each eye. With the retinoscopic reading she stated she could see nothing with the right eye.

It seems impossible to fix the cylindric error. It would appear that her trouble with the right eye was due to ciliary spasm. I should greatly appreciate a suggestion in such a case.

A. E. W.

Answer.—Analysis of the case presented by the correspondent shows the following: For the right eye the sphere varied from +0.25 to —1.5 (retinoscopic and subjective tests) and the cylinder from +0.50 to —0.75, with the axis at or close to 90 degrees for the plus cylinder and at 180 degrees on the only occasion when a minus cylinder was recorded. For the left eye the sphere varied from —0.12 to +0.50 and the cylinder from —0.50 to +0.87.

The correspondent suggests that the discordant results may be due to undue accommodative tension (ciliary spasm). If this is the case, a search should be made for physical defects, such as menstrual disorders, blocking of the nasal sinuses, contact points between the turbinates and the septum, other nasal pathologic involvement, focal infection and emotional upsets.

If such defects are nonexistent or, if present, have been corrected, it is suggested that an ocular examination be made with a view to answering the following questions:

1. Does the corneal surface present any irregularities as shown by the biomicroscope or the Placido disk?

2. Is the iris tremulous?

3. Is the anterior chamber of equal depth throughout? (If not, is the lens tilted, or is there a partial backward dislocation?)

4. Is there any evidence of metamorphopsia?

5. Is there any contraction of the form field?

6. Is there any evidence of paracentral scotoma (tangent screen or stereocampimeter)?

It is assumed that a searching ophthalmoscopic examination has failed to reveal any pathologic involvement, either in the central or in the peripheral parts of the eyeground.

It should be recalled that every eye possesses some irregular astigmatism, which may modify the regular variety. The irregular astigmatism is not measurable by retinoscopic examination, though its presence may be suspected by the queer behavior of the shadow. It is present when the patient, suitably fogged, gives confusing—and to the examiner, disconcerting—answers when exposed to an astigmatic chart, e. g., when the 60 and 110 degree lines are declared to be equally distinct.

The following procedure is recommended:

1. Have the patient instil a 1 per cent solution of atropine into each eye three times a day for at least two weeks.

2. Do a retinoscopic examination at least twice at two days intervals, having the patient continue to use the atropine. If the results are not comparable, have the patient continue to use the atropine for another week.

3. The subjective tests should be begun with the eyes considerably overcorrected (vision reduced to 20/60 by plus spheres).

4. With a suitable astigmatic chart (Lancaster or Verhoeff), determine the approximate axis of the astigmatism. Display a crossed bar chart (Camp Thomas is suggested) at the selected axis and use minus cylinders at right angles to this axis until the cross bars are equalized.

If there is doubt about the exact position of the cylinder axis, use the crossed cylinder (0.37 or 0.50) for axis, keeping the eye moderately fogged (vision not over 20/30). Reduce the sphere slightly and check the cylinder strength by using the crossed cylinders for strength, not only with test letters but with a suitable crossed line dial.

Give nearly full correction and insist on the patient's wearing glasses constantly. During the patients recovery from full cycloplegia, have her use a weak solution of atropine (0.1 per cent) for at least two weeks.

A heart to heart talk with the patient (and also her mother) may secure the cooperation necessary to carry out this strenuous program. But if the case is a genuine one of ciliary spasm, which seems probable, any less drastic procedure will probably prove ineffective.

After omission of the atropine, encourage the patient to put up with the blurred vision and have her report at weekly intervals to prove to her the improvement in distant sight as the ciliary spasm relaxes.

The successful management of such a case is a tribute to the "drive" of the examiner. He must get the patient's full confidence, as her cooperation in wearing the glasses constantly (despite the initial distance fog) is vitally necessary if comfort is finally to be attained.

BREAK-RESISTING LENSES

To the Editor:—In a drive to insure protection against injuries to the eyes, my co-workers and I have been using break-resisting lenses, known as super-armor-plate. The question has come up as to whether the flat lens or the toric is preferable in the plano. P. R. L.

Answer.—Two types of protective lenses are in use: a laminated one which consists of two pieces of glass with a plastic material cemented between them and a heat-treated or hardened lens which under dropping ball tests will crack radially but will not chip if in a frame. These lenses come in a six base curve and a flat type, the curved, or toric, lens having the greater fracture-resisting property. This advantage is based on the principle set forth in physics that an arched bridge is much stronger than one laid on pillars or abutments.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Biochemistry

THE REACTION AND BUFFER ACTIVITY OF NORMAL OX LENSES. P. W. SALIT, Am. J. Ophth. **22**: 413 (April) 1939.

Salit gives the following summary of his investigations:

"The hydrogen-ion concentration and the buffer activity of normal calf and two-year-old cattle lenses were studied electrometrically, using the hydrogen electrode and a saturated calomel cell. In this study it was found that if a single lens is emulsified in 15 cc. of distilled water, the dilution, on account of the high buffer property of the lens substance, produces practically no change in the hydrogen-ion concentration. The average p_H of calf lenses is 7.56 and that of two year old cattle is 7.43. The latter figure is in exact agreement with the p_H value found by Sauermann for normal cattle lenses by the Michaelis colorimetric method in which precautions were taken to prevent the loss of carbon dioxide. It is therefore concluded that normal cattle lenses contain no free carbon dioxide."

W. S. REESE.

THERMOELECTROLYTIC COAGULOFLOCCULATION OF AQUEOUS EXTRACTS OF THE CRYSTALLINE LENS (NORMAL AND CATARACTOUS). A. RUBINO, Ann. di ottal. e clin. ocul. **67**: 389 (May) 1939.

The author had previously investigated in various ocular diseases the flocculation of serum in aqueous solution when exposed to an electrolytic current (reaction of Weltmann). He now reports studies on extracts of the crystalline lens with respect to the concentration of electrolytes combined with heat necessary to produce flocculation in aqueous extracts of the normal and the cataractous lens. The aqueous solutions employed did not coagulate with heat alone but only when electrolytes of a definite concentration were added. The "electrolytic threshold" thus determined for extracts of rabbit, beef and human lens was: for calcium chloride, a concentration of 0.08 to 0.05 per cent, and for sodium chloride and potassium chloride, 0.6 to 0.3 per cent. The threshold was lower for extracts of the nucleus than for extracts of cortex or of the whole lens. The threshold for extracts of naphthalene and traumatic cataract in the rabbit and of senile human cataract was much higher than that of the normal lens (0.4 to 0.1 per cent for calcium chloride). The aqueous of normal animals behaved like solutions of electrolytes in producing with heat flocculation of normal lens extracts. This was true also of the aqueous of animals with experimental cataract. Flocculation did not occur, however, when aqueous was added to extracts of the cataractous lens, the absence of the reaction depending, apparently, on some chemical change in the lens resulting from the cataractous process. One reason for the higher threshold of the cataractous lens is apparently the smaller amount of soluble protein in such lenses. Another

factor, the author believes, is the increased amount of lipids, especially stearin, in the cataractous lens, which has a protective effect on the soluble proteins, preventing flocculation.

S. R. GIFFORD.

Conjunctiva

CULTIVATION IN VITRO OF HUMAN CONJUNCTIVAL AND CORNEAL EPITHELIUM. P. THYGESON, Am. J. Ophth. **22:** 649 (June) 1940.

Thygeson gives the following summary and conclusion of his study of human conjunctival and corneal tissues as culture mediums for the viruses of trachoma, inclusion blennorrhea and molluscum contagiosum:

"The cultivation of human conjunctival and corneal epithelium in vitro is reported, with notes on the histology of the developing tissues. A technique is described which is believed to be suitable for use in the study of such viruses as may require human epithelial tissues for propagation."

W. S. REESE.

Cornea and Sclera

METASTATIC SEPTIC ENDOPHTHALMITIS WITH RING ABSCESS OF THE CORNEA. C. W. TOOKER, Am. J. Ophth. **22:** 526 (May) 1939.

Tooker discusses endogenous infection of the eye and reports a case of metastatic ophthalmia six months after an operation for mastoiditis, the patient dying of septicemia three days after admission to the hospital. The autopsy and pathologic reports on the affected eye are given, and the following summary is made:

"All the changes in the eyeball are secondary to the ocular primary foci in the retina and the ciliary processes. Emboli of bacteria must first have lodged in the capillaries of the retina and in those of the ciliary processes. It is unusual to have two distinct primary foci in the same eye, thus involving the retinal and ciliary vascular systems, although but a very small part of the latter is concerned. From the capillaries of the retina and ciliary body the bacteria easily enter the vitreous chamber. Here, by chemotactic action, they produce infiltration of the retina and to a small extent of the ciliary body with pus cells and the exudation of pus cells from the ciliary body, retina, and optic papilla into the vitreous. The ring abscess of the cornea and necrosis of the iris are, of course, secondary to toxins coming from the vitreous. As Axenfeld pointed out, a post-mortem increase of the bacteria would seem to be the most probable explanation of their large number within the eye."

W. S. REESE.

CONGENITAL SYPHILIS WITH DEVELOPMENT OF INTERSTITIAL KERATITIS AFTER TRAUMA: REPORT OF TWO CASES. N. GORCHAKOVA and I. GORCHAKOV, Vestnik oftal. **15:** 94, 1939.

In 2 children, aged 8 and 9 years, whose Wassermann reactions and also those of their mothers were 4 plus, parenchymatous keratitis developed in one eye after trauma. A review of the literature on the subject is given. The Gorchakovs accept the Vignol-Lutati theory that trauma weakens the local immunity of the cornea and aids proliferation of the previously latent Spirochaeta pallida.

O. SITCHEVSKA.

Experimental Pathology

FACTORS IN FUNCTIONAL RECOVERY FOLLOWING SECTION OF THE OCULOMOTOR NERVE IN MONKEYS. M. B. BENDER and J. F. FULTON, J. Neurol. & Psychiat. **2**: 285 (Oct.) 1939.

In man, after monolateral paralysis of the oculomotor nerve the eyelid of the affected side fails to move down in unison with the lid of the normal side and may actually move up (the "pseudo Graefe" sign). Similar abnormal movements have been reproduced experimentally in monkeys and chimpanzees, and the evidence points to misdirected regeneration as the cause of the dissociated palpebral and ocular movements, supporting the views expressed by Bielschowsky. In all the animal studies some recovery of nerve function occurred after section of the third nerve.

J. E. LEBENSOHN.

METABOLISM OF THE RETINA AFTER COMPRESSION OF THE GLOBE. A. DE CRECCHIO, Ann. di ottal. e clin. ocul. **67**: 739 (Oct.) 1939.

Compression of 100 Gm. was applied to the eyes of rabbits with the Bailliart dynamometer for periods of thirty to one hundred and eighty minutes, and the retinas were studied in the Warburg microrespiratory apparatus. Anaerobic glycoylsis was decreased only in the eyes compressed for one hundred and eighty minutes. Oxygen consumption was decreased in eyes compressed for sixty minutes, while in those compressed for ninety and one hundred and eighty minutes it was increased. The decrease in glycolysis and increase in oxygen consumption after the longer periods of compression indicate degenerative changes as a result of the induced ischemia, with freeing of substances such as fats from the degenerated tissues.

S. R. GIFFORD.

ALKALINE RESERVE OF THE BLOOD AND p_H OF THE AQUEOUS IN EXPERIMENTAL ACIDOSIS. A. DE CRECCHIO and D. BRIGNOLA, Ann. di ottal. e clin. ocul. **67**: 781 (Oct.) 1939.

The literature is reviewed, with a table listing previous findings. Details of technic are discussed which seem to account for some of the conflicting results reported. In the authors' work the albino rabbit was employed, as acidosis is easily produced in this animal. The alkaline reserve of the blood was determined by the Van Slyke method at intervals during the production of acidosis by intravenous injection of acids or by administration of acids by stomach tube combined with dietary control. Determination of the p_H of the aqueous was made by the electrolytic method with a hydrogen electrode, the fluid being tested in a syringe with a platinum tip and copper wire embodied in the plunger. Determinations were done under a carbon dioxide tension of 47.7 mm. of mercury. Control animals with a carbon dioxide tension in the blood of 48.5 and 49.2 per cent showed p_H values of 7.49 to 7.55 in the aqueous. In animals with acidosis in which the carbon dioxide tension of the blood dropped to 32.4 and 24.8 per cent, the p_H of the aqueous showed a progressive reduction, being, on the average, 7.28 during acidosis. Figures as low as 7.11, 7.14 and 7.16 were observed in some animals. This change in p_H of the aqueous, as compared with the absence of any change in p_H of the blood except in extreme acidosis

incompatible with life, shows that the buffer system of the aqueous is much less effective than in the blood, while the parallelism between reduction of carbon dioxide tension in the blood and p_H of the aqueous is evidence that the aqueous is an ultrafiltrate of the blood. A bibliography is included.

S. R. GIFFORD.

General Diseases

RELATION OF HYPERGLYCEMIA AND HYPOGLYCEMIA OF THE BLOOD TO INTRAOCULAR TENSION. THE INFLUENCE OF NORMAL AND HYPOGLYCEMIC SERUM ON THE OCULAR TENSION OF THE RABBIT. V. CAVKA, Ann. d'ocul. **176:** 616 (Aug.) 1939.

The majority of the patients whom the author studied presented hypoglycemia in the course of a state of coma. He cites 2 cases of dementia praecox seen at the neurologic clinic and gives the results of his study in tabular form. The period of observation for the patients, who had been given a large dose of insulin, was five days. The ocular tension was taken at about 11 o'clock in the morning with the patients in a state of coma with serious symptoms and in a complete state of hypoglycemia; it was taken again in the afternoon when the hypoglycemic shock had lessened. In these 2 cases there was found hypotonia during the height of the reaction, dilatation of the retinal veins and slow reaction of the pupils with alternate miosis and mydriasis. Blepharospasm seemed to be a constant symptom.

The author also gives the details of 20 cases of dementia praecox in which insulin therapy was used following the method of Sakel. Observations were made twice during increased hypoglycemia in the morning and twice in the afternoon.

The experiments that the author afterward undertook were based on the influence of hypoglycemic serum and normal serum on the ocular tension. He tried the effect of conjunctival injections of insulin on the blood vessels of the retina, on the pupillary reaction and on the ocular tension. The results are given in tabular form.

S. H. MCKEE.

THE OCULAR SYMPATHETIC-PARASYMPATHETIC SYSTEM IN EXOPHTHALMIC GOITER. E. TROVATI, Ann. di ottal. e clin. ocul. **67:** 757 (Oct.) 1939.

Ten normal persons and 9 with exophthalmic goiter were tested with cocaine and epinephrine. The group of patients showed an exophthalmos which was unilateral or more marked on one side. Cocaine in 2 to 5 per cent solution and epinephrine in a dilution of 1:1,000 were instilled and in some cases injected subconjunctivally. No significant changes were observed in the pupillary reaction of the patients with exophthalmic goiter as compared with normal persons. Pupillary dilatation was greater, however, on the side showing least exophthalmos in patients with predominantly unilateral exophthalmos. The effect of cocaine in enlarging the palpebral fissure and increasing the exophthalmos was also more marked on the side showing least exophthalmos. No differences were observed in the two groups in the effect of the drugs on the conjunctival vessels. The miosis which followed mydriasis in all cases shows that these drugs stimulate not only the sympathetic but the parasympathetic system.

S. R. GIFFORD.

Glaucoma

CONGENITAL SECONDARY GLAUCOMA. A. V. HALLUM, Am. J. Ophth. **22**: 1262 (Nov.) 1939.

Hallum reports the cases of 2 Negro infants whose corneas were cloudy and lusterless and whose ocular tension was elevated. Trephining was done on one eye of the first child, but both eyes later cleared up under antisyphilitic therapy. In the second case the eyes cleared up without the aid of a miotic. Hallum concludes that when an infant has glaucoma syphilis should be suspected and intensive treatment instituted before an operation is considered.

W. S. REESE.

THE UNDERLYING CAUSES OF GLAUCOMA, INCLUDING NOTES ON THE LINES OF ENQUIRY WHICH HAVE BEEN PURSUED, WITH SUGGESTIONS AS TO FUTURE RESEARCH IN CLINIC AND LABORATORY. P. J. EVANS, Brit. J. Ophth. **23**: 745 (Dec.) 1939.

This study received the Middlemore Prize for 1939.

The author gives a historical survey of the causes of glaucoma, together with recent theories. The hypothesis put forward in this essay is that the cause of primary glaucoma is capillary and venous stasis and that the condition arises from changes resulting from such a disturbance of the local circulation. It is held that the symptom complex known as glaucoma includes a number of separate conditions whose only features in common are those represented by ultimate decompensation of the mechanics provided for the excretion of fluid from within the eye. It will be shown that primary glaucoma, or glaucoma simplex is the local manifestation of a general disease, or, in the words of Lagrange, that "the eye attacked by hypertension is a sick eye in a sick body and not merely a hypertonic organ but also a sclerosed and dystrophic organ that shares the trouble with the rest of the body."

The author admits, however, that there are a number of predisposing and contributory factors whose importance must be duly estimated: the proportionate size of the lens to the eye as a whole; smallness of the hypermetropic eye; hypertrophy of the ciliary muscle and the steadily increasing size of the lens as life advances; the increasing sclerosis of the pectinate ligament with advancing years, and exfoliation of the lens capsule.

These changes may provide an additional factor favoring the onset or maintenance of glaucoma in predisposed eyes but cannot be considered as causes per se. Closure of the angle of the anterior chamber is a secondary result of the glaucoma, though a complication of great importance. Loss of elasticity of the sclera itself is probably not of great importance apart from the possible pressure on the vorticose veins passing through it.

The author believes that the course of further inquiry into glaucoma must therefore be directed toward the elucidation of the precise mechanism of those factors influencing the state of the optic nerve, and, when possible, toward the control or correction of disease processes when present.

The article is illustrated.

W. ZENTMAYER.

DIATHERMIC PUNCTURE OF THE CILIARY BODY: A NEW OPERATION FOR GLAUCOMA. H. WAGNER and H. RICHNER, Schweiz. med. Wchnschr. **69:** 1048 (Oct. 28) 1939.

On the basis of a statistical analysis of the results of treatment in cases of glaucoma at the ophthalmologic clinic of Zurich from 1936 to 1939, Wagner and Richner demonstrate the great importance of a new method devised by Vogt—diathermic acupuncture of the ciliary body. After thorough anesthesia and exposure of about 2 cm. of the superior or inferior or of the temporal or nasal half of the sclera, diathermic acupuncture is begun with a current of from 50 to 60 milliamperes and with a needle which is 0.5 mm. in length and has a caliber of from 0.15 to 0.18 mm. With this needle, from two to fifteen diathermic punctures are made in a beltlike arrangement 3 mm. away from the limbus. Each puncture requires one-half second, and the distance between the punctures is from 0.5 to 1 mm. In the same manner an equal number of diathermic punctures are made 4 mm. away from the limbus, and, if necessary, the same number are made at a distance of 5 mm. From some of the punctures a watery or a stringy fluid exudes, and there is a considerable reduction in tension. As soon as the bulb is soft, the operation is terminated by suture of the conjunctiva. It is important that the diathermic punctures be made not too close to the limbus, because this involves the danger of corneal and lenticular injuries. The pains which develop several hours after the operation can be controlled by antineuralgic remedies. The statistical report presented by the authors reveals that this method of treatment was followed by normal pressure in 72.3 per cent of the cases. This figure is high when it is considered that in the majority of cases one of the customary operations had failed or had been unsuitable. The authors used to perform the classic operations in cases of acute and primary glaucoma and of secondary glaucoma after iridocyclitis, but by means of diathermic puncture of the ciliary body they are now able to cure glaucoma in a large number of cases in which it was formerly incurable. The operative procedure is not difficult technically, and it can be repeated several times without danger.

W. ZENTMAYER. [J. A. M. A.]

Injuries

PHENOL BURNS OF BOTH EYES WITH GENERAL SEQUELAE: COMMENT ON THE THERAPY OF CAUSTIC BURNS OF THE EYES; REPORT OF A CASE. A. WINKLER, Klin. Monatsbl. f. Augenh. **102:** 810 (June) 1939.

A mixture of phenol containing 35 per cent of cresol and 10 per cent of xylenol splashed into both eyes of a man aged 33. Caustic burns of the third, the necrotizing, degree of the eyes, face and chest followed. Chemosis, clouding of the corneas, petechial hemorrhages in the conjunctiva and iritis resulted; vision was reduced to perception of motion of the hand. Transient diffuse nephritis and severe but transient anemia developed. This was the result of the toxic action of the phenol absorbed by the organism. Conservative treatment was administered, and vision of 5/25 of the normal was regained in the right eye and in the left eye, of 5/7 of the normal. It consisted in the removal of every trace of the caustic burns, irrigation of the eyes with saline solution every

hour or two, applications of an ointment containing vitamin A (vogan) and one containing ethylmorphine hydrochloride and instillation of mild mydriatics, such as homatropine and scopolamine. No bandage was applied. This therapy has been used at the ophthalmic clinic of the University of Halle for 215 patients during the last five years.

K. L. STOLL.

Instruments

ADHESIONS OF THE IRIS DURING OPERATIONS: AN INSTRUMENT FOR REPOSITION OF THE IRIS. W. COMBERG, Klin. Monatsbl. f. Augenh. **102:** 44 (Jan.) 1939.

Adhesions of the iris may cause complications not only during operations for cataract and glaucoma but also in cases of penetrating injuries of the eyeball. Adhesions of the iris following operations may be divided into three groups: those that occur after iridectomy, peripheral circular adhesions which occur during extraction of a cataract and adhesions of the iris to the upper lip of the corneal incision which occur after divers operations in which the eyeball is entered. The author discusses these three groups in detail and describes methods devised to prevent complications following the adhesions. For replacing the iris and separating the adhesions, he uses a small instrument of his own device. It consists of a small ball, attached to a handle, with a short point protruding from the center of the ball. The ball will prevent injuries to the tissues adjoining the adhesions.

K. L. STOLL.

Lens

THE DISLODGING FORCE UTILIZED IN INTRACAPSULAR CATARACT EXTRACTION. T. J. DIMITRY, Am. J. Ophth. **22:** 416 (April) 1939.

Dimitry believes that intracapsular extraction is accomplished by pushing rather than pulling the lens and that it is important that this be understood. He states that the vitreous maintains itself better when the lens is pushed out.

W. S. REESE.

CONGENITAL CATARACTS AND OXYCEPHALY. G. P. SOURDILLE, E. DUVAL and GENDRON, Bull. Soc. d'opht. de Paris **51:** 186 (March) 1939.

In a recent thesis Lartigue has shown the rarity of congenital cataracts associated with oxycephaly. Two observations are cited. A girl of 7 years of age was born at six and one-half months. A cranial deformity was noted. Vision had always been poor. Both lenses had lamellar cataracts. The hands and feet had the classic aspect of arachnodactylia. The roentgenograms of the skull showed signs of hypertension with digital impressions on the convexity of the skull. Examination of the fundi after operation for the cataracts failed to reveal any change from the normal.

The second patient was 5 years of age. The family history showed no hereditary stigmas. The patient was thought to have normal vision as a baby. In later years it was found to be deficient. Lamellar cataracts were discovered. A roentgenogram of the skull was char-

acteristic of oxycephaly. After operation for the cataracts the papillae were found to be edematous with congested borders. Pictures and roentgenograms are included.

L. L. MAYER.

MECHANISM OF EXPULSIVE HEMORRHAGE. J. VAN DER HOEVE, Arch. f. Ophth. **139:** 655 (Oct.) 1939.

In expulsive hemorrhage a choroidal vessel bursts, and the blood spurts into the subretinal space and causes the retina to tear. Therefore, fragments of retina are found among the tissues that plug the external wound. The average frequency of expulsive hemorrhage during or following cataract extractions is 0.2 per cent.

P. C. KRONFELD.

Lids

SPASTIC-ENTROPION CORRECTION BY ORBICULARIS TRANSPLANTATION. J. M. WHEELER, Am. J. Ophth. **22:** 477 (May) 1939.

Wheeler describes two methods of correcting spastic entropion. With the first, he transplants a strip of orbicularis muscle, about 4 mm. wide, from the outer half of the lid to the periosteum at a point almost level with the outer canthus. The second method consists of a tucking or shortening of a strip of orbicularis muscle, about 4 mm. wide, just below the tarsus of the lower lid. This shortened strip is sutured to the tarso-orbital fascia about 2 mm. below the tarsus.

W. S. REESE.

BLEPHAROCHALASIS. H. G. HADLEY, Acta ophth. **17:** 310, 1939.

As originally described by Fuchs, blepharochalasis is characterized by recurrent, progressively more frequent attacks of transient edema of the upper eyelids, beginning in early life and resulting in marked atrophy and permanent baggy swelling of the skin.

The author records a case with these typical features.

O. P. PERKINS.

Methods of Examination

PRACTICAL IMPORTANCE OF ARTERIOGRAPHY IN OPHTHALMOLOGY: REPORT OF CASES. A. VOGT, Klin. Monatsbl. f. Augenh. **102:** 641 (May) 1939.

Vogt renders a short account of the history and technic of encephalo-arteriography, practiced first by Moniz and then by Santos, Lamas and Caldas. This method compares favorably with ventriculography after the method of Dandy. Three cases in which arteriography was successfully carried out by the brain surgeon Krayenbühl are reported. This method was employed by him in a total of 40 cases, in none of which were adverse results observed. The arteries in the brain are made visible by opacification with a solution of colloidal thorium dioxide which is injected into the carotid artery. The use of sodium iodide has been abandoned since 1930, as it had caused too many fatalities. Arteriography is done while the patient rests on the roentgen table during the seconds, or fractions thereof, during which the thorium dioxide passes through the carotid artery. Between 6 and 8 cc. are used for the purpose

in Zurich. The conditions in the 3 cases reported by Vogt were: (1) a large aneurysm of the internal carotid artery, located in the middle cranial fossa; (2) an aneurysm, smaller than in the first case, in the S-shaped curve of the right internal carotid artery, which pressed on the oculomotor and trigeminus nerves, and (3) beginning traumatic pulsating exophthalmos in a girl aged 9 years. In the third case an iron rib of an umbrella had entered the orbit, passed into the cavernous sinus and torn a hole into the internal carotid artery. Aphasia, hemiparesis and pulsating exophthalmos disappeared after arteriography, and vision and the visual field became normal.

K. L. STOLL.

ROENTGENOGRAM OF THE SKULL IN CALCIFICATION OF THE INTERNAL CAROTID ARTERY AND OTHER OCULAR DISEASES: REPORT OF CASES. M. GOEDE, Klin. Monatsbl. f. Augenh. **102**: 651 (May) 1939.

The author took roentgenograms of the skull of 120 patients aged 50 or more years. They suffered from senile cataracts, glaucoma without tension, arteriosclerosis of the choroid with general hypertension, defects of the field of vision without definite causation, glaucoma, tabetic or traumatic simple atrophy of the optic nerve, optic neuritis, choked disk and tumors of the hypophysis. One of 4 patients without ocular disease showed bony changes in the sella turcica. Calcification of the internal carotid artery was present in one third of these patients, mostly associated with other cerebral calcifications or with minor osseous changes in the area of the sella turcica. No calcification of the carotid artery was found in 46 per cent of patients with severe general arteriosclerosis, especially of the retinal vessels. Hence, arteriosclerosis of the basal cerebral arteries need not be associated with that in the peripheral, or retinal, arteries. Mere compression of the optic nerve by the calcified carotid artery need not produce atrophy or functional disturbances of this nerve, as evidenced by the roentgenograms; disturbances of the circulation, however, may cause lesions of the optic nerve. The roentgenogram is important in the diagnosis of all obscure lesions of the optic nerve or in those connected with arteriosclerosis or in vascular disease.

K. L. STOLL.

Neurology

SYPHILITIC OPTICOCHIASMATIC ARACHNOIDITIS. D. VAIL, Am. J. Ophth. **22**: 505 (May) 1939.

Vail discusses basilar syphilis and reports 7 cases of syphilitic opticochiasmatic arachnoiditis. He gives the following summary and conclusions:

"The literature on syphilitic opticochiasmatic arachnoiditis is reviewed, and seven additional cases, six of which are presumptive, are reported. Three of these cases were diagnosed by neurologists as tabes or taboparesis. A history of vascular lesions was obtained in two cases. The fields of vision showed defects that are considered to be characteristic of chiasmal involvement. Attention is directed to the mixed type of optic atrophy seen in all cases. The writer considers this type as one of the pathognomonic signs of chiasmatic arachnoiditis, and that it indicates a combination of simple atrophy and neuritis.

"This study indicates that if neuro-surgery is performed before it is too late improvement in vision may result. Hans Reese, neurologic editor of the 1937 Year Book of Neurology, Psychiatry, and Endocrinology, in commenting on Hausman's paper, says that: 'The more acute syndromes of this entity should be treated medically prior to surgical intervention, whereas a damaged optic chiasm with progressive field defects (low-grade reactivity in blood and spinal fluid) should be treated at first surgically and thereafter medically.'' W. S. REESE.

OCULAR DISTURBANCES OF HYPOPHYSIAL ORIGIN IN THE COURSE OF EXOPHTHALMIC GOITER (BITEMPORAL NARROWING OF VISUAL FIELDS). F. SCHOUSBOE and SARROUY, Bull. Soc. d'opht. de Paris **51:** 191 (March) 1939.

For the past two years the authors have observed a patient with hyperthyroidism and leukomelanoderma. In spite of medical treatment, a cardiac condition ensued, and the basal metabolism progressively increased. Surgical intervention was proposed, but a bitemporal loss of visual fields made the authors think of the possibility of a disease of the diencephalon in view of the pigmentary disturbance previously reported by Risak (*Wien. klin. Wchnschr.* **50:** 14, 1937).

In addition, it was found that the patient had increased in weight from 43 Kg. in 1936 to 58 Kg. in 1938. A roentgenogram of the sella turcica was negative. Roentgen therapy was given to the hypophysial area. Under its influence the symptoms cleared rapidly. There was, however, a slight complete loss of the bitemporal fields of vision, which persisted. The theories of the causative factor are interesting. Charts of visual fields and a bibliography are included. L. L. MAYER.

ARTERIAL RETINAL TENSION IN INTRACRANIAL TUMORS: ITS IMPORTANCE FOR TOPOGRAPHIC DIAGNOSIS AND ITS ROLE IN FORMATION OF CHOKED DISK. G. DEMORSIER, M. MONNIER and E. B. STREIFF, Rev. neurol. **71:** 702 (June) 1939.

DeMorsier and his co-workers review the evidence pertaining to 21 cases of intracranial tumors, verified as such surgically or at necropsy, in order to determine the significance of cerebral tumors for retinal and spinal tension and the formation of choked disk. The ages of the patients ranged between 11 and 60 years. The authors established two indexes: a retinohumoral tension index, obtained by dividing the normal diastolic retinal tension by the normal diastolic humoral tension of 21 patients, and a retinospinal tension index, ascertained by dividing the retinal tension by the spinal tension. Methods of measurement and arithmetical results are indicated. They found (1) that, contrary to accepted views, intracranial tumors were accompanied much more frequently by retinal hypotension or normal retinal tension than by retinal hypertension (retinohumoral hypertension was present in only 6 of the 21 cases, or 28 per cent, hypotension in 12 and normal tension in 3) and (2) that the retinohumoral tension index unmistakably indicated variations according to the site of the cranial tumor, with greater retinal hypertension accompanying tumors in the posterior cerebral fossa (adverse conditions in 2 cases). From these observations, the authors deduce that other factors besides brain compression are productive of retinal hyper-

tension (frequently observed, e. g., in sequels of craniocerebral traumas). Retinal tension may well be conditioned by retinal disturbances in the control centers of cerebral vasomotricity. In their study of retinospinal tension in relation to choked disk, the authors were led to form the following observations: 1. The retinospinal tension index is not affected by the position of the tumor. 2. A certain connection exists between spinal tension and choked disk formation, papilledema invariably arising when spinal tension rose to 80 or higher but being capable of developing with spinal tension as low as 40. 3. Choked disk appeared (with two exceptions) when the retinospinal tension index fell below 0.58 (below half the normal rate of about 1.2). The authors think that these observations, which they admit need to be studied more extensively, have interpretative significance for noncerebral tumors. They point out that choked disk is much less frequent in cases of intracranial tumors than is generally assumed. The low frequency (8 times in 21 cases, or 38 per cent) is ascribed by them to the regular employment of encephalography.

J. A. M. A.

So-Called Permanent Choked Disk: Report of Cases. F. Fischer, Klin. Monatsbl. f. Augenh. **102**: 656 (May) 1939.

Pick claimed in 1904 that choked disk may exist unchanged for years in consequence of increased cerebral pressure. Some authors accepted this contention; others again doubted it. Fischer describes 2 cases in support of Pick's claim. Increased cerebral pressure was produced by a tumor of the hypophysis and a hemangioma, respectively. Choked disk existed in the first case for five years at least and in the second case for over fourteen years. Vision remained good in each case.

The causative disease in these cases appeared to be benign; nevertheless, the author cannot explain why the fibers of the optic nerves could escape destruction by edematous changes which last for so many years.

K. L. Stoll.

Ocular Muscles

Concomitant Strabismus Among Negroes at Gabon, French Equatorial Africa. S. Holm, Acta ophth. **17**: 367, 1939.

Among 1,931 Negroes at Gabon, French Equatorial Africa, the author found 15 cases of strabismus, in 10 of which there was no other ocular pathologic involvement. Among these 10, alternating convergent strabismus occurred once and alternating divergent strabismus 9 times. Thus the incidence of strabismus among Negroes seems much lower than among white persons, the difference apparently being the result of the extreme rarity of convergent strabismus in the Negro. The incidence of divergent strabismus in the two groups does not seem to differ much.

The author explains the rarity of convergent strabismus in the black race on the basis of the low incidence of any high degree of hyperopia and hyperopic astigmatism among them. Moreover, they show anisometropia much less frequently than do white persons—a factor which explains the predominance of alternation in the black race. The divergent character of the squint in these Negroes is probably explainable on the basis of a low refractive error together with a large biorbital angle.

O. P. Perkins.

Orbit, Eyeball and Accessory Sinuses

FUSO-SPIROCHETAL INFECTION OF THE EYE AND ORBIT. G. L. WALKER, Am. J. Ophth. **22:** 622 (June) 1939.

Walker cites the case of a 47 year old white woman who stated that four weeks after the extraction of a right upper molar, the socket of which continued to drain, redness and swelling of the eyelids developed on the same side and gradually increased. Examination revealed an anemic, emaciated, white woman, confined to bed with far advanced generalized atrophic arthritis. The mucous membrane of the mouth appeared healthy, but in the socket of the first right upper molar was a fistulous tract leading into the antrum. A probe passed through was covered with foul-smelling, purulent discharge. The lids of the right eye were red and edematous. A profuse, yellowish white, foul-smelling discharge exuded from the palpebral fissure. There was moderate proptosis, and movements of the globe were limited. The palpebral conjunctivas of both lids were covered with pseudomembranes, which on removal revealed a marked papillary hypertrophy. Smears from the conjunctiva and oral fistula revealed large numbers of spiral and fusiform organisms. Forty-eight hours after the patient's admission to the hospital a perforating ulcer of the conjunctiva and sclera developed at the lower limbus. On the ninth day the eye was a purulent necrotic mass, and a probe was easily passed from the orbit into the antrum and out through the tooth socket. Nine days after her admission symptoms of meningitis developed, and she died two days later. Necropsy revealed basal meningitis which appeared to have extended from the orbit along the optic nerve. The immediate cause of death was a large subdural hemorrhage arising from a ruptured aneurysm of the left internal carotid artery just proximal to its bifurcation. Smears from various infected areas showed fusiform and spiral bacteria with the morphologic structure of Vincent's organisms.

W. S. REESE.

Parasites

ECHINOCOCCOSIS OF THE ORBIT. R. R. WARSHAVSKAYA, Vestnik oftal. **15:** 82, 1939.

Two cases of echinococcosis of the orbit are reported. One case was that of a 17 year old student who had pain and bulging of the right eye for one month. There was marked exophthalmos, with edema and redness of the upper lid and papilledema of the fundus. A fistula was situated at the external canthus, with stringy matter discharging from it, which was found to be fragments of an echinococcus cyst. An incision after the method of Golovine was made, and the cyst was removed in fragments. Ten days later mastoiditis developed on the right side which necessitated anthrotomy.

The second patient, aged 24, had a gradually developing exophthalmos for one year with impairment of vision and hearing and a change in his mental behavior. The exophthalmos and papilledema were marked; a small tumor was palpated at the upper wall of the orbit. The left frontal lobe was involved, and the middle ear was inflamed. An echinococcus cyst with three small cysts included were found on operation.

A few months later the mental symptoms disappeared, but the vision was poor because of atrophy of the optic disk.

The special features of these cases were: the formation of a fistula and the development of mastoiditis in the first case and involvement of the frontal lobe and otitis media in the second.

A detailed review of the literature is given. Of 191 cases reported from 1915 to 1937, 44 echinococcus cysts were reported in the Russian literature. Warshawskaya believes that Uhlenhuth's test of the blood should be done, as a positive reaction is specific for echinococcus cyst.

O. SITCHEVSKA.

Pharmacology

MYDRIATIC ACTION OF ADRENOXIN. P. HEIRMANN and M. GOFFART, Compt. rend. Soc. de biol. **132:** 84, 1939.

Adrenoxin, an oxidation product of epinephrine, is a powerful and lasting mydriatic, ten thousand times as effective as epinephrine. Adrenoxin, 1 : 100,000, is about equal in mydriatic activity to atropine in a dilution of 1 : 100.

J. E. LEBENSOHN.

EFFECTS ON THE EYE OF A NEW IMIDOAZOLIN (PRISCOL-CIBAQ). M. GLEES, Arch. f. Ophth. **140:** 497, 1939.

Priscol, a product of the Swiss firm Ciba, is an imidoazolin which is related to histamine and to pilocarpine. On instillation of the aqueous solution of the drug into the conjunctival sac of experimental animals, the vessels of the conjunctiva and iris become markedly dilated and the aqueous cloudy. At the same time the pupil dilates. The intraocular pressure remains unaltered. On explanted iris muscles, the drug paralyzes the sympathetic nerves. Considering the close chemical relation of the drug to pilocarpine and histamine, its dilating effect on the pupil is remarkable.

P. C. KRONFELD.

EFFECT ON THE INTRAOCULAR PRESSURE OF AN EXTRACT FROM THE IRIS AND CILIARY BODY. B. BOROS, Arch. f. Ophth. **140:** 507, 1939.

A protein-free extract from the iris and ciliary body of pigs' eyes was found to have a fairly regular effect on the tension of normal and of glaucomatous human eyes. The intramuscular or intravenous injection of the extract was followed at first by a slight elevation and then by a more marked depression of the intraocular pressure. This diphasic reaction was more pronounced in glaucomatous than in normal eyes. The injection of the extract also lowered the blood pressure by about 10 mm.

P. C. KRONFELD.

Retina and Optic Nerve

DISCIFORM DEGENERATION OF THE POSTERIOR POLE OF THE RETINA. B. ADAMANTIADIS, Ann. d'ocul. **176:** 537, 1939.

Disciform degeneration of the posterior pole of the retina, first described a few years ago, is known under a number of different names. In the French literature it is called senile macular exudative retinitis, while in the German literature it is known as disciform macular degeneration. Recently Rintelen described it under the name of senile macular pseudotumor.

The author chose the term disciform degeneration of the posterior pole of the retina because it refers to cases in which the lesion is localized at the posterior pole, without always involving the macula. It is closely related to two other diseases of the posterior pole, exudative retinitis of Coats and circinate retinitis of Fuchs, with which it is frequently confounded by many authors.

Three cases are described in detail which are similar except in the minor points enumerated. Three illustrations and numerous references accompany the article.

S. H. McKee.

Examination of the Fundus of the Eye During Blood Transfusions (Venous Tension). I. Borsotti and P. Pillet, Ann. d'ocul. **176:** 586, 1939.

In this study the authors noted especially the retinal circulation in relation to the general tension in the course of blood transfusions. Their attention was attracted particularly by changes observed in the central vein of the retina.

They noted two different kinds of reaction to transfusion. In a certain number of patients the general tension showed a tendency to rise; in the same patients there was observed the disappearance of a spontaneous venous pulse without any alteration in the retinal veins.

In another category of patients in whom, on the contrary, the general tension shows a tendency to be lowered, it was observed that where the venous pulse appeared or where it had been present there was a definite increase in the pulsation of the central vein.

After discussing the probable causes of the phenomenon, the authors conclude that probably it is due to a diminution of the venous tension, which may be an expression of passive or active dilatation of a certain part of the venous network as opposed to an increase of the blood mass. This interpretation, although completely hypothetic, is in accord with the observations of certain authors who have already noted a diminution of the systolic beat and a minute beat (cardiac) and a diminution of the blood circulation at the same time as an important increase in the total blood mass. One may, in effect, in these cases, attribute to the venous network the function of an active or passive reservoir.

Seven tables are shown, and a bibliography accompanies the article.

S. H. McKee.

Tumors

Melanosarcoma of the Iris. W. B. Doherty, Am. J. Ophth. **22:** 239 (March) 1939.

Doherty discusses sarcoma of the iris and reports 3 cases. He draws the following conclusions:

"1. From the histories of these tumors it is reasonable to suppose that they are less malignant than those of the choroid or ciliary body. This may be due to the fact that they are more easily observed, and immediately operated upon.

"2. A beginning malignancy of a melanoma of the iris, as viewed from a clinical standpoint, would manifest itself by (1) an increase in size; (2) varying discolorations in the same growth; (3) an increased blood supply; (4) elliptical pupil and development of tension.

"3. A cyst and a melanosarcoma may co-exist.

"4. The so-called ring sarcomas are extensions around the iris from the primary growth.

"The question whether pigmentation plays a role in the malignancy of these growths is a much disputed one. I believe it does. A deeply pigmented growth with pigment running wild must mean something. It is true that we do have malignant leucosarcomas, but do they metastasize very rapidly, and do they not occur most frequently in the young?

W. S. REESE.

Uvea

CLINICAL AND SEROLOGIC DIAGNOSIS OF GONORRHEAL IRITIS. E. J. SIEGERT, Arch. f. Ophth. **139:** 303, 1939.

Typical gonorrheal iritis is an acute metastatic disease characterized by sudden, severe irritation of the affected eye, severe pain and pronounced loss of vision. There is profuse gelatinous exudation into the anterior chamber which accounts for the tendency toward formation of multiple synechiae. These, however, can be broken easily by mydriatics. Iris nodes are absent. The patients give a history of gonorrheal infection which usually dates back several years. Gonorrheal arthritis preceded the iritis in only 3 of Siegert's 11 cases. At the time of the iritis it is often difficult to detect definite gonorrheal foci in the urogenital tract. The serologic tests, that is, the complement fixation or precipitin tests in which various antigens prepared from gonococci are used, are fairly reliable and of definite diagnostic value. especially since extraocular gonorrheal foci may not be detectable at the time of the iritis.

P. C. KRONFELD.

OPERATIVE THERAPY IN TUBERCULOUS IRIDOCYCLITIS. W. HALLERMANN, Klin. Monatsbl. f. Augenh. **102:** 318 (March) 1939.

Hallermann reports on the operative treatment of tuberculous iridocyclitis and its frequent complication, secondary glaucoma. His report is based on observations on about 1,350 persons treated at the tuberculosis sanatorium at Höchenschwand, Black Forest, Germany. The results obtained with iridectomy were discouraging. The more exudative the condition of the eye, the more unfavorable the prognosis. An exact iridectomy is imperative; the pillars must be smooth and free from impaction. Even proliferating and fibrous inflammation may flare up after iridectomy. The author disputes the curative result of this operation claimed by some writers in tuberculous cases; on the contrary, he found only 9 of 65 eyes cured on which iridectomy had been performed elsewhere prior to admission of the patients to the sanatorium; late recurrences were observed even after perfect operation. Glaucoma persisted in 20 per cent of iridectomized eyes.

Far better results were obtained with transfixation after E. Fuchs's method. Hallermann performed this operation on 4 patients at the sanatorium and on 18 at the ophthalmic clinic of Freiburg. In 5 of these patients the operation had to be done during fulminant attacks of glaucoma. The results were good in every case but 2, in which the opening of the transfixation closed after twenty-four and thirty-six

hours, respectively. Iridectomy was performed in these 2 cases afterward, with satisfactory result. A double-edged knife, 4 mm. wide, is used for the transfixation. A lesion of the lens was avoided in each case. Fuchs's technic of cutting the fibers of the iris perpendicularly to their course is advisable. Recurrences were not observed, but data regarding late results could not yet be gathered. K. L. STOLL.

Therapeutics

EFFECT OF ULTRAVIOLET RAYS ON NORMAL HUMAN EYES AND EYES WITH SOME CORNEAL DISEASES. E. TROVATI, Ann. di ottal. e clin. ocul. **67:** 481 (July) 1939.

The author employed the arc lamp of Birch-Hirschfeld and the mercury vapor lamp of Bach, the latter being apparently employed in standardizing the dose and in most of the therapeutic work. With it, exposure for ten minutes at 5 cm. produced only slight hyperemia in 2 of 10 cases, while exposure of fifteen minutes produced fairly marked and moderately painful reactions after six hours, which lasted twenty-four hours or longer. Exposures of twenty and twenty-five minutes produced somewhat more severe reactions, but these subsided without any permanent damage to the eye. Succeeding doses, slightly larger, given after intervals of two days produced less reaction than was produced by single doses of the same duration. Such tolerance to increasing doses was observed in all experiments and indicates that subsequent doses must be increased in order to produce the same reaction as that caused by the initial dose.

In cases of corneal disease doses increasing from three to thirty minutes were administered, with the object of producing repeated moderate reactions. These cases included 5 of scarring from ulcerative keratitis, 2 of interstitial keratitis, 5 of vascular pannus and 2 of disciform keratitis. The local reactions, accompanied by increase in the vascularity previously present, resulted in some absorption of the accompanying corneal opacity. While vision remained unchanged in some cases, it improved considerably in others, in 1 case from 1/60 to 2/10. In the 2 cases of disciform keratitis, the opacity was completely absorbed. From six to nine doses at intervals of two days to two weeks were given in most cases. The effect of irradiation on intraocular tension was noted in a number of cases. A moderate increase of tension was observed, proportional to the duration of exposure. It appeared immediately after treatment and began to recede after six hours, and tension dropped to a figure below the initial one, as a rule, in twenty-four hours. A bibliography is included. S. R. GIFFORD.

Society Transactions

AMERICAN OPHTHALMOLOGICAL SOCIETY

E. V. L. Brown, M.D., *President*

Eugene M. Blake, M.D., *Secretary*

Seventy-Sixth Annual Meeting, Hot Springs, Va., June 3-5, 1940

Sterilization of Sharp Instruments by Chemical Solutions. Dr. M. Hayward Post, St. Louis.

The subject is considered from two standpoints: (1) the proper sterilization of instruments and (2) the preservation of instruments during sterilization. A number of solutions originally suggested by the late Dr. A. E. Ewing twelve years ago are described. Experimental work done at that time by Dr. Post to determine the sterilizing properties of these compounds was reported in the *American Journal of Ophthalmology* (**11:** 18, 1928) and is briefly reviewed in this paper. The results obtained with these solutions during the past twelve years are also presented. In addition, new solutions are described and a few of the commercial preparations now on the market briefly discussed. The experimental work was done at the Oscar Johnson Institute.

DISCUSSION

Dr. W. B. Lancaster, Boston: Dr. Post, in his abstract at least, said nothing about the objections to boiling. I have been an advocate of boiling for many years but have not done anything recently in the way of research. I should like to call attention to the danger of tarnishing or possibly of oxidation. I am not sure about the chemical change which may occur from boiling. About forty-five years ago, when I first joined the staff of the Massachusetts Eye and Ear Infirmary, I asked the nurse in the operating room to let me boil my instruments. She said "No, it spoils the instruments," and in my cocksure way I said, "Oh, no, I have boiled them many times, and it does not hurt them." So she placed a new cataract knife in the water in the sterilizer, to which had been added sodium bicarbonate. After being boiled in the sterilizer, the cataract knife was gray and tarnished. The nurse had proved her point. I said, "You cannot boil instruments in that sterilizer, but I will bring one in which they can be boiled." I got an enamelware container and boiled some knives and various other steel articles for many hours without any of them tarnishing. We examined the first sterilizer. It was made of tinned copper, the inside being the tin surface, from which a good deal of the tin had worn off, and the bottom, which had evidently leaked at times, had been repaired with large quantities of solder, so that all around the bottom was a heavy layer of solder. We then had what Dr. Post speaks of as alloys; the lead, tin and copper and the steel of the knife in the boiling alkaline solution evidently produced the tarnishing. After we used the enamelware dish or sterilizer we did not have any trouble.

At that time knives were made with bone handles; later they were made with handles of aluminum and various kinds of metals. If a knife with an aluminum handle is boiled long in an alkaline solution, the handle will be dissolved, aluminum being soluble in alkalis but not in acids.

However, it must be emphasized that it is not necessary to boil instruments a long time to sterilize them. When we were working on that problem I took a number of small machine screws, because they were rough and bacteria could be pretty well protected in the crevices, and dipped them into a mixed culture of various bacteria, then dried them. For the test they were boiled for five, ten, twenty and thirty seconds, and for one, five and ten minutes. After being boiled each screw was dropped into a tube of culture medium. The tubes were turned over to Dr. Verhoeff, who was in charge of the laboratory of the infirmary. To his surprise, and much to my surprise, when the tubes were examined in a couple of days the mediums in all except one were turbid. Even the medium containing the screw that had been boiled for ten minutes was turbid. The culture medium that was not turbid happened to contain a brass screw, and the other mediums contained steel screws. Dr. Verhoeff was enough interested to examine cultures to see what organism could have grown and made the medium turbid in spite of that treatment, so he made subcultures; every one of them was sterile. The turbidity was simply due to oxidation of the steel screw; in other words, five seconds sufficed to sterilize every one of those specimens. It is unnecessary to boil a knife five minutes or even one minute. What I do is have the nurse dip the knife for fifteen to twenty seconds into boiling water containing sodium bicarbonate; such a procedure is more effective—far more effective—than putting the knife in alcohol for ten or fifteen minutes. Of course, Dr. Post's solution is far more effective than alcohol. I think it is an advantage if one can get away from the use of these solutions. Perhaps that idea is a personal idiosyncrasy. I feel such confidence in the effectiveness of boiling that I still adhere to it as a good method, while freely admitting that Dr. Post's method is also good.

Dr. Sanford R. Gifford, Chicago: May I ask Dr. Post whether he uses formaldehyde or solution of formaldehyde in his solution 3?

Dr. John Green, St. Louis: Dr. Post's solution no. 1 has been in use in the ophthalmic clinic and operating room of the St. Louis City Hospital for a number of years. All cutting instruments are immersed in this solution for fifteen minutes; all others are boiled. The majority of the ocular operations have been cataract extractions, many on extremely old, poorly nourished and decrepit persons. The hospital has an excellent record of freedom from postoperative infections—evidence of the efficiency of sterilization with chemicals and heat.

Instruments freshly sharpened by the instrument maker are often returned to the surgeon coated with oil or petrolatum. I have been surprised to observe that some surgeons and nurses place these oil-coated instruments directly in the chemical sterilizer. Is there any assurance that the chemical penetrates the oily film and reaches the surface of the knife or scissors? It has been my practice to remove all oily film by a thorough washing in hot soapsuds, with a final rinse in sterile water before immersion in the chemical.

DR. M. HAYWARD POST, St. Louis: I have the greatest regard for anything that Dr. Lancaster has to say. In an abstract of his paper, which appeared in the ARCHIVES OF OPHTHALMOLOGY (**30**: 394, 1901), I believe I said "It is well known that knives may be boiled in water, especially in alkaline solutions, provided no electrolytic action takes place." In the recent experimental work reported the knives were boiled in dishes of pyrex or bakelite. Apparently the difficulty arises in the composition of the handles. I do not believe that the average ophthalmologist will know enough concerning their composition to make boiling a safe method of sterilization. Either the knife blade or the handle seems to have been injured in most of the experiments, so I feel that this is a precarious procedure for the average ophthalmologist. Of course, much shorter periods of boiling are more practical than those I used, which are fifteen minutes or longer. The first three knives (shown for inspection) were boiled months ago and at that time were in good condition, but when they were examined a short while ago the blade of the third knife had taken on a bluish appearance and had lost its edge to some extent.

As to the use of alcohol alone, Dr. Ewing determined that 95 per cent alcohol, after a period of three weeks, produced considerable rust on wire nails.

In reply to Dr. Gifford's question about the formaldehyde component, solution of formaldehyde U. S. P. is used. It contains 37 per cent of formaldehyde gas in aqueous solution.

I was interested in what Dr. Green said about the solution being used at the St. Louis City Hospital. I did not include it in my list, because I was not absolutely certain of the facts concerning its use in that hospital at this time.

There is a slightly disagreeable odor to solution no. 3, but it does not cling long and one is not supposed to wash one's hands in it. For office practice I have added oil of lavender, but I did not conduct my experiments with this component present, so I cannot speak of the sterilizing effects of such a solution at this time. I will say that solutions no. 1 and 2 are the least injurious to the knives after exposure for long periods of time.

Anterior Scleral Staphyloma and Retinal Detachment Cured by Excision. DR. DERRICK VAIL, Cincinnati.

On June 15, 1936, a woman aged 57 suddenly noticed markedly blurred vision in the left eye. It was found to be reduced to ability to see hand movements in the lower temporal field. A large detachment of the retina was discovered in the upper temporal portion. On June 30 the conjunctiva was dissected from the upper temporal portion of the eyeball and an area of well circumscribed ectasia of the sclera, measuring about 12 by 12 mm., was disclosed. Transillumination gave negative results. Two double-armed sutures were inserted along the edges of the staphyloma and looped out of the way. A fixation suture was inserted in the middle of the area of ectasia. The major portion of the staphyloma was excised with elliptic incisions, with resultant loss of fluid vitreous. The main vitreous body was not lost. The sutures were tightly tied, the edges being brought closely together. The eye healed satis-

factorily, and the retina adhered in place. There was slow but steady improvement in vision. On Oct. 27, 1939, the vision was 20/40 +, and the patient could read Jaeger's test type 2 with correction. The field of vision was full. Ocular tension has remained normal.

DISCUSSION

DR. H. M. LANGDON, Philadelphia: There is one phase of Dr. Vail's case which I think opens up a rather interesting question. To be sure, the condition in his case is not actually comparable with ordinary detachment of the retina. His patient presented definite bulging of the sclera, and the sclera separated from the retina rather than the retina from the sclera, as occurs in the case of ordinary detachment. During the operation Dr. Vail necessarily made a large tear in the retina; yet in spite of that tear, which he did nothing to close other than the mere excision and removal of the bulge with replacement of the sclera in its original position, the retina went back into place and presumably has stayed there for three years. This opens up a question as to whether tears in the retina are as serious and have as much to do with separation of the retina as one sometimes thinks. The effect of the tear on the retina and the question of detachment are to me interesting points which are not settled by a long way. I think that one should make every effort to close tears, but I am not sure that a tear is as serious a factor in starting detachment as is sometimes thought, as is demonstrated by Dr. Vail's case.

DR. DERRICK VAIL, Cincinnati: I am sure that I cannot answer some of the questions raised by Dr. Langdon's discussion except to say I do not know exactly how the retina was severed. In talking it over with Dr. Verhoeff we thought that since the retina was so intimately connected with the staphylomatous process on the posterior portion (that is, toward part of the equator), the blades of the scissors must have caused some trauma to the rest of the retina when the staphyloma was excised. Examination of the retina on the tenth or twelfth day after operation, when one could get a good view of it, showed a wavy white edge, which no doubt was the edge of the retina, and the appearance was just about the same as that of a retina following diathermic coagulation; in other words, there was a whitish, edematous, elevated surface.

Death Following Operation for Cataract: Report of Three Cases. DR. CHARLES M. SWAB, Omaha.

A general consideration of the subject is presented, and the brief life expectancy of many patients with cataract is pointed out. Certain hazards for elderly persons are discussed. The case histories of 3 persons who succumbed after cataract operation are detailed. The first patient died of streptococcic meningitis; the second, of hypostatic pneumonia, and the third, of intestinal obstruction.

DISCUSSION

DR. HANS BARKAN, San Francisco: I should like to add briefly a report of 5 cases in which death occurred after cataract extraction in the

last five years in combined private and clinical practice, just to emphasize Dr. Swab's statement that old persons are sometimes not as safe a risk for what is often considered a minor surgical procedure as the physician and the family assume them to be. Four of the patients were over 70; the other was aged 64. Autopsy was permitted in 3 of the 5 cases.

In the first case autopsy revealed thrombosis of the superior mesenteric artery with infarction of the small intestine, thrombosis of the renal artery with infarction of a kidney, mural thrombosis of the coronary artery and general and local arteriosclerosis of the coronary arteries.

The final diagnosis in the second case, that of a woman aged 70, was bronchial pneumonia, acute parotitis on the left side, senility, arteriosclerosis with hypertension, arteriosclerotic heart disease and auricular fibrillation. Autopsy was not performed.

The patient in the third case, a woman aged 73, had a blood pressure of 210 systolic and 110 diastolic; otherwise there were no particular abnormalities. The day after operation she became confused and nauseated and vomited; as she was dehydrated, fluids were forced by vein and by clysis. Four days after operation she died, presumably of acute auricular fibrillation. Autopsy was not performed.

The patient in the fourth case, a man aged 78, entered the clinic with glaucoma. An Elliot trephine operation was performed. The patient progressed smoothly for three days after operation, when he had a sharp pain in the chest and right jaw; there was no dyspnea. Four hours later, while washing, the patient died. Autopsy showed pulmonary embolism and thrombosis of the periprostatic and the right femoral veins.

The patient in the fifth case, a woman aged 77, entered the clinic for a cataract extraction. Autopsy showed acute gangrenous cystitis, acute pyelonephritis, multiple renal abscesses, metastatic carcinoma in the uterus and lymph nodes in the pelvis. In this case it was known that the patient had these conditions and that complications might arise. Her two sons begged to have the operation performed, so that the care of their blind old mother would be somewhat easier for them.

Death in the first 4 cases was due to heart failure and might have happened at any time instead of after cataract extraction.

Dr. C. A. Clapp, Baltimore: In discussing Dr. Swab's paper I desire to report 2 cases. The first is that of a man of 80, who was sent to the hospital by his nephew, who was a leading medical man of Baltimore and who reported that the patient was a normal risk for his age. However, on the fifth day a vascular cerebral accident developed and the patient died suddenly. In the second case the patient, a man aged 58, had locomotor ataxia but otherwise was a fairly satisfactory risk. An acute cardiac condition developed on the third day after operation. It was found at that time that the patient was in the habit of taking large quantities of alcohol. In spite of supplying him with alcohol thereafter and having a cardiac specialist treat him as scientifically as possible, he died from acute cardiac failure.

Dr. Arnold Knapp, New York: I should like to speak of the second point which Dr. Swab raised: the question of hypostatic pneumonia after cataract extraction. I think the present after-treatment of cataract has

reduced the frequency of this complication; in fact, according to Elschnig it has been eliminated. At the same time, there are still patients in whom bronchopneumonia develops, and some of them die; the type of pneumonia is not hypostatic and to my mind is due to a definite infection of the respiratory tract. I therefore suggest that operation for cataract on elderly persons should be done at a time of year when infections of the respiratory tract are not prevalent and that the possibility of infection be guarded against by limiting the visitors; if there is any doubt, not only the visitors but the physicians and nurses should wear protecting masks.

Dr. Burton Chance, Philadelphia: I want to take exception to the title of Dr. Swab's paper. From the histories which he relates it strikes me that the deaths were incidental; they did not immediately follow, nor were they directly associated with, the cataract operation. So commonly is one asked by the family if the patient's operation is dangerous, that if they saw such a title as "Death Following Operation for Cataract" they would naturally be skeptical as to the prognosis. I have in mind 2 patients whose deaths I have believed were due to emotion after the extraction. One was a woman crippled with kyphosis; she died purely from emotion late in the afternoon after the extraction. She had been blind for many years, and the joy of seeing again was so great she could not stand it. The other patient, a Civil War veteran, was the postmaster in his little town. He had been blind for a great many years; all the work of the office was carried on by a member of his family. I operated on him, and the result was entirely satisfactory; glasses were prescribed within ten days. The joy of going home again and seeing the hills of his country was too much for him. As his niece opened the cab door on taking him home, he fell dead in her arms. I look on that death as one due to emotion following cataract extraction.

Dr. Luther C. Peter, Philadelphia: Apropos of the third cause of death to which Dr. Swab referred, I should like to say that paralytic ileus is a common condition in cataract extraction. I have never had a resulting death, but I have had 3 instances of paralytic ileus, 2 of which occurred in the same patient. Through the help of a surgeon, we immediately established the Wangensteen method of duodenal drainage, and the patient recovered. I have seen a number of these cases before I saw the 3 referred to, which I think were cases of early paralytic ileus. If one inquires into the history of these cases, it is possible to find, in many instances, evidence in previous years of the condition which is spoken of as paralytic ileus.

Dr. Charles M. Swab, Omaha: I do not believe that any ophthalmologist anticipates a death from cataract extraction or he would not assume the responsibility of the operation. On the other hand, I think that in a good many of the cases in which death occurs after operation and in which one does not have the benefit of autopsy that the diagnosis is probably inaccurate. I was never exactly satisfied with the case of hypostatic bronchopneumonia, because in spite of the fact that it was felt that everything had been done to prevent the patient's getting hypostatic pneumonia it did develop. A long while ago, in fact in the beginning of this century, the majority of deaths that were reported after cataract operation were practically all due to diabetic coma. Of course

that misfortune does not happen any more. It is interesting to see how many deaths nowadays are associated with heart disease. However, this is not necessarily true with regard to patients with cataract.

Removal of the Lens in Cases of High Myopia in Which Lenticular Opacities Prevent Improvement of Vision by Correcting Glasses.
Dr. Burton Chance, Philadelphia.

Surgeons in the past century, dreading complications, hesitated to extract the lens in cases of high myopia. Discission and other absorptive measures were often fatal. Patients with myopia seen by the author in his early days are compared with those examined in later years. Moderate changes in the lenses have little significance in low degrees of myopia but may cause great diminution of sight in high degrees. In the author's experience the removal of the crystalline lenses in such cases has been performed with safety and has effected marked improvement in vision.

DISCUSSION

Dr. Allen Greenwood, Boston: My experience with such cases up to within a few years has not been very encouraging. I found that when I did a combined extraction that I was likely to have considerable lens cortex left and to have to do a discission. I was loath to try extraction in the capsule in eyes with high myopia, but finally I concluded that the zonule of such eyes must be rather feeble and that I might attempt it. Since then I have had some exceptionally good results with this type of extraction. I have had no bad after-results and have obtained excellent vision.

I have 1 patient of middle age for whom I prescribed a — 32 D. lens for constant wear. With a — 6 D. sph. over it she obtained excellent vision for the theater and similar places. Measured with a concave retinoscope, she had about 35 to 38 D. myopia. She had lost one eye, the left one, and a cloudy lens developed in the right eye; her vision was so reduced that she had to be practically led around. I took out the lens in the capsule without loss of vitreous, and I think she is one of the most grateful patients I have ever had. She can see well without a glass, though she has a little better vision with a minus spherical lens. She is the only patient I have ever had to whom I had to give a minus lens after operation. With the combination of a minus sphere and a plus cylinder she can read Jaeger's test type 2.

I have had a number of patients with myopia of over 20 D. for whom I have taken the lens out with the capsule. In none of these cases has there been any loss of vitreous. In the case of high myopia of 38 D. the lens was extracted almost entirely by traction with the capsule forceps with almost no pressure exerted from below. I want to recommend that instead of a combined extraction and cutting of the capsule the lens be extracted in the capsule.

Dr. F. H. Verhoeff, Boston: I agree with Dr. Chance completely in his view that the lens should be removed in such cases, and I agree with Dr. Greenwood's views about intracapsular extraction. I have been doing this type of extraction in such cases for a good many years. Many

of the patients have prominent eyes, so that according to the current views loss of vitreous should be feared. However, this has not deterred me, and I have had less loss of vitreous in these cases than is customary. There are certain other things I have found which have not been mentioned in regard to these cases. I have noticed that the patients do not get good vision for an unusually long time after operation. At the end of six weeks the patient may have vision of only 20/200 and yet six months later have vision of 20/40 or better. I do not know why this is. I suppose edema of the macula develops which interferes with vision and that it takes some time for it to disappear. I have also found that these patients have much more astigmatism than others. Some have 4 or 6 diopters. At first I was afraid that separation of the retina might occur afterward, but this has not been my experience. I think Dr. Haskett Derby used to say that if there is marked myopic chorioretinitis, separation of the retina will not occur in cases of high myopia. I know that is not strictly so, but I think there is a great deal in it.

Dr. Chance's paper does not deal with the question of the young myopes with clear lenses. I have removed the lenses of several such persons by discission followed by linear extraction with good results, but it seems to me if one can get reasonably good vision with glasses it is a little safer not to do the operation.

As Dr. Greenwood says, these patients are the most grateful ones an ophthalmologist has. They often see better after the operation than they have ever seen, and they are much impressed by the fact that they can see well without glasses. I tell them they are lucky to have a cataract, because otherwise I might not have felt like operating on them.

Dr. Luther C. Peter, Philadelphia: I do not want to repeat the discussion which was so well given by Dr. Greenwood and Dr. Verhoeff. My experience runs exactly parallel. I simply want to add, however, that one can make such intracapsular operations perfectly safe by resorting to the use of avertin with amylene hydrate and also by the use of a corneoscleral suture, combined sometimes with conjunctival sutures, if indicated.

Dr. Burton Chance, Philadelphia: I read in abstract the histories of but 2 of the cases included in my report. In all of them there may be found some of the details on which Dr. Verhoeff commented, namely: the later visual improvement and the fact that I deemed it unnecessary to extract the lenses in their capsules. In 1 instance I did a true extraction and brought the lens out with the forceps. In none was there escape of vitreous, nor has there been a subsequent detachment of the retina; in 1 case there has developed a cyst of the retina, far down in the nasal quadrant. This has remained exactly as when I first saw it. The patient refuses operation, as she has had fairly good vision which she does not want to risk.

Discovery of a Neuromyoarterioglomus Tumor in the Eyelid. Dr. Daniel B. Kirby, New York.

This article will be published in full, with discussion, in a later issue of the Archives.

Psammoma of the Orbit. Dr. W. E. Fry and Dr. P. DeLong, Philadelphia.

This report concerns the clinical signs and pathologic changes in a case of primary tumor of the right orbit. The patient was a 32 year old white man with a history of a painless exophthalmos of the right eye for three years. The right globe was pushed down and out. Roentgen examination revealed destruction of the upper and inner walls of the orbit. At operation a partial cystic tumorous mass was found in the upper inner angle of the orbit which had involved the right frontal sinus. No intracranial connection was found, and the tumor mass was entirely outside of the muscle cone. Microscopic examination showed that the more solid parts of the tumor mass consisted of whirls of elongated cells of scanty cytoplasm and elongated nuclei. The centers of many of the whirls were occupied by psammoma bodies. The appearance was similar to that of an intracranial meningeal fibroblastoma.

DISCUSSION

Dr. Allen Greenwood, Boston: I want to call the attention of the authors to the publication about forty-two years ago by Dr. Munro, of Boston, who at my suggestion operated for an orbital tumor in the lower part of the orbit which was pushing the eye up and out. It was necessary before Dr. Munro got through the operation to extirpate the whole of the right upper jaw. Dr. F. Mallory examined the tumor and pronounced it a psammoma of the antrum and the orbit.

Dr. W. E. Fry, Philadelphia: I believe in reviewing the literature that I saw the article to which Dr. Greenwood refers. As I recall, there is an illustration of the patient and also a photomicrograph which corresponds pretty accurately to the one I have shown here. In referring to these cases I did not necessarily mean they were the only ones in the literature. I do not think there are a great many, but I have tried to pick out reports which indicated tumors of this type in various locations.

Scotoma Associated with the Menstrual Cycle. Dr. John N. Evans, Brooklyn.

This report presents evidence of the evolution of a scotoma of the central field which is plottable shortly before the menstrual flow appears and which enlarges to maximum size and then disappears before the flow has entirely ceased.

The studies are arranged in groups and bring together a series of maps which were made during the menstrual flow and a series which were made during the intermenstruum. Scotometric readings were taken at daily intervals in order to follow the evolution of the defect and to compare it with known events of the menstrual cycle.

A brief review of the physiology of menstruation is set forth and an effort is made, in graphic form, to show the variations of the scotoma as related to phases of the menstrual cycle.

There seems to be a relation between the defect and the well recognized accumulation of estrogenic substances and the increased permea-

bility of systemic capillaries. Since the defect represents an inferior, or dependent, retina and since it is obviously an angioscotoma, it seems reasonable to infer that it arises from increased permeability of the retinal capillaries or at least from transitory circulatory insufficiency.

Reference is made to visual field defects during the menstrual period as reported by other workers.

DISCUSSION

DR. CONRAD BERENS, New York: I was fortunate in having the test objects and exact technic Dr. Evans employs in studying these patients. I have been able to find the scotoma of menstruation in 3 of 4 young women examined. The fourth showed no scotoma, at least not with the tangent screen; the other women, who showed the scotoma on the stereocampimeter with a 0.4 mm. test object, showed a similar scotoma with the tangent screen.

It appears from limited studies that possibly some of the patients may not exhibit the scotoma of menstruation.

When the neutral gray tangent screen was used at 750 mm., it was necessary to use a 0.5 mm. test object in order to bring out the scotoma associated with menstruation which is demonstrable with the stereocampimeter with an 0.4 mm. test object. I was unable to study the fields of any pregnant woman.

DR. JOHN N. EVANS, Brooklyn: In order to interpret Dr. Berens' negative result it would be necessary to have supplementary data on blood pressure and other clinical data.

Sulfanilamide Therapy of Inclusion Conjunctivitis. DR. PHILLIPS THYGESON, New York.

This article will appear in full, with discussion, in a later issue of the ARCHIVES.

Convalescent Blood for Herpes Zoster: Second Report. DR. TRYGVE GUNDERSEN, Boston.

Ninety-one patients with herpes zoster ophthalmicus have now been studied. In 61, or 66 per cent, ocular infections developed. A total of 22 of these were treated with convalescent blood. The results are compared with those obtained in a control series of 39 patients whose eyes were affected but who received no specific therapy. This study confirms the previous conclusions regarding the value of treatment of herpes zoster ophthalmicus with herpes convalescent blood. If between 250 and 400 cc. is given before ocular infection is well established, good results may be expected.

DISCUSSION

DR. FRANCIS HEED ADLER, Philadelphia: I should like to ask Dr. Gundersen how long immunity persists if a patient has had herpes; that is, whether it persists throughout his life. Would it be possible to have the blood kept in a blood bank? Would the immune properties persist? It is not always easy to find a patient who has recovered from herpes zoster when one needs the blood.

I should also like to ask Dr. Gundersen if he has had any experience with the use of smallpox vaccine in cases of herpes zoster.

Dr. Trygve Gundersen, Boston: I do not know how long immunity lasts in herpes zoster, but it probably lasts a long time—perhaps throughout life. Immunity seems to develop early. I had an excellent result by treating a patient with blood from a donor who had had herpes zoster only three weeks before. I have had no experience in using blood from blood banks.

I have never used smallpox vaccine in cases of herpes zoster, but I have used it in cases of recurring herpes simplex and dendritic keratitis. Ten patients with recurring dendritic keratitis were vaccinated approximately seven years ago. Already 2 of them have had recurrences.

Dr. Francis Heed Adler, Philadelphia: I was not referring to the use of smallpox vaccine in the prevention of ocular symptoms but to the treatment of such symptoms once they have appeared. It has been advocated by a number of ophthalmologists that vaccination with smallpox vaccine has a favorable influence on ocular involvement.

Accidental Involvement of the Eyes in Vaccinia. Dr. A. O. Pfingst and Dr. C. D. Townes, Louisville.

The complications, local and constitutional, which may arise from vaccination against smallpox are briefly considered. The frequency of accidental infection of the eyes and their adnexa is relatively high and on the basis of recent reports would seem to be higher than earlier statistics indicated. The infection is carried to the eye from the vaccinated area by the hands. Strangely enough, only 25 per cent of cases are the result of autoinfection, transmission usually occurring through the hands of the mother or of other attendants. The conjunctiva seems to be the most vulnerable tissue, although cases have been recorded in which the skin of the lids was first involved. Corneal infiltration or true pustular corneal involvement occurs in about 1 of every 10 cases.

Contrary to what might be expected, direct transmission from the vesicles of vaccinia of the cow to the milker is almost unheard of. Diligent search in the American and the foreign literature revealed only a single case. One such occurrence was observed in a dairyman who had milked cows during an epidemic of vaccinia and had carried the infection to his eyes with his fingers.

DISCUSSION

Dr. Walter S. Atkinson, Watertown, N. Y.: This subject should be of some interest to ophthalmologists, because the percentage of corneal involvement that I discovered in looking over the literature for the past twenty years is a little different from that reported here. In 91 cases of vaccinia with ocular involvement reported, the cornea was involved in 32. Therefore there is possibly some impairment of vision in approximately a third of the cases. The clinical picture along with the history is quite characteristic, so that a diagnosis can usually be made with reasonable certainty. A few years ago when I saw a case of vaccinia of the lids I was unaware of the fact that there were laboratory tests to

confirm the diagnosis, and in case there may be members who are also unaware of that fact, may I say that about fifteen years ago a complement fixation test and an agglutination test were reported to confirm the diagnosis of vaccinia and smallpox?

DR. F. H. VERHOEFF, Boston: I should like to ask Dr. Pfingst and Dr. Townes if in any of these cases they tried to confirm the diagnosis by infecting the cornea of a rabbit with the secretion. It can be infected with vaccine virus.

DR. J. S. FRIEDENWALD, Baltimore: Had the patient in the case reported here previously been vaccinated?

DR. A. O. PFINGST, Louisville: I will answer the last question first. The patient, who was 70 years of age, had not been vaccinated in recent years. He was vaccinated when 12 years old and had not recently been exposed to the vaccinia of man. He had always been robust and healthy.

In reply to Dr. Verhoeff, no study was made of this question. We did make bacteriologic examinations of the secretion from the eyelids and found the white staphylococcus. I went out to the farm to try to get a specimen from the vesicles of the cow but found that the condition had run its course and that no more vesicles were present.

Keratoconjunctivitis Sicca. DR. GORDON M. BRUCE, New York.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

Leptotrichosis Conjunctivae (Parinaud's Conjunctivitis) Without Glandular Involvement: Report of a Case. DR. F. H. VERHOEFF, Boston.

This paper will be published in full in a later issue of the ARCHIVES.

Site of the Disturbance of Tonic Pupils (Adie's Syndrome). DR. HAROLD G. SCHEIE AND DR. FRANCIS HEED ADLER, Philadelphia.

Eleven patients with tonic pupils were studied for their reaction to various drugs. All showed marked sensitivity to acetylbetamethylcholine (mecholyl), a cholinergic drug. The results led to the hypothesis that tonic pupils could be due to a partial denervation of the postganglionic parasympathetic fibers to the sphincter.

A series of cats were operated on; one ciliary ganglion was removed from some of them, and the postganglionic fibers of others were crushed. All the cats showed sensitivity to acetylbetamethylcholine in concentrations which had no effect on the normal pupil. Section of the preganglionic fibers failed to cause sensitivity. Regeneration of the postganglionic fibers which had been crushed led to a recovery of function; during one stage of the recovery the pupillary reactions were sluggish to light, although typical tonic pupils were not produced.

A tonic pupil was seen postoperatively in 1 case of retinal detachment in which extensive electrocoagulation with Walker needles was done. Sensitivity to acetylbetamethylcholine developed in this eye. In a second case of retinal detachment a similar sensitivity to the drug

developed postoperatively. This was believed to be due to damage done to the long ciliary nerves. These facts tend to support the foregoing hypothesis.

Other patients with known postganglionic parasympathetic lesions were given acetylbetamethylcholine and found to exhibit sensitivity. These patients had conditions which included: (1) orbital tumor with involvement of the third nerve, (2) increased intraocular pressure and (3) old iritis with anterior synechia.

The rationale for the use of acetylbetamethylcholine in cases of glaucoma is considered on this basis. The use of this drug to differentiate tonic pupils from Argyll Robertson pupils is advocated.

DISCUSSION

Dr. Edward Jackson, Denver: While perhaps the 2 cases that I wish to mention do not strictly come under the heading of tonic pupils, they suggest an approach and a side of the subject which one ought to bear in mind.

A mining engineer a little over 30 years of age came to me with the history that when he went down a shaft of 500 or 600 feet with three or four other men, on reaching the bottom of the shaft the other men could immediately see in the reduced light but that he could see nothing. It was ten or fifteen minutes and sometimes almost half an hour before he recovered his sight, and then he could see as well as the others. I examined his eyes and found absolutely no reaction to light in either pupil. The pupils were small, between 2 and 3 mm. habitually, and could not be dilated by any mydriatic.

A woman came to the clinic after a number of mydriatics instilled by different persons at different times over a period of two weeks or more failed to dilate the pupils beyond 2 mm. in diameter, although in every other respect the eyes seemed to be normal. It was necessary to measure the refraction as well as could be done with a 2 mm. pupil, and glasses were given that were satisfactory.

The best explanation for these 2 cases seems to be that the original structure of the iris was such that it was incapable of reaction to light. I have seen only these 2 cases; such cases are probably extremely rare, but they should be borne in mind. The condition in both of these was bilateral and was probably due to the essential development of the eye. This limitation of the subject of tonic pupils may be guessed from such cases.

Dr. Francis Heed Adler, Philadelphia: The experimental and clinical data presented indicate that the site of the lesion is in the third nerve, somewhere between the ciliary ganglion and the nerve endings in the sphincter muscle, and that this lesion partially destroys these fibers. The result is a pupil which is permanently sensitized to cholines—either those produced normally in the tissues of the body or those introduced experimentally.

In cats the experimental lesions produced sensitized pupils which were definitely sluggish in their reactions when the damage to the nerves was partial, but we were not able to produce typical tonic pupils.

In man, however, we unwittingly have produced typical tonic pupils.

We have seen sensitization to acetylbetamethylcholine in cases of old iritis in which presumably the nerve endings in the muscle might have been partially damaged.

In a number of cases of chronic glaucoma sensitivity occurred even though the tension had been normalized by a trephine operation. In these cases we believe that the previous increased tension caused partial damage to the nerve fibers.

In every case of glaucoma in which the tension is elevated sensitivity is found. This makes acetylbetamethylcholine a valuable drug to use in the treatment of acute glaucoma. In these cases, however, as soon as the pressure has been lowered the sensitivity is lost.

In several cats the intraocular pressure was artificially raised in one eye and sensitivity was produced on this side.

The diagnostic value of acetylbetamethylcholine in differentiating tonic pupils from Argyll Robertson pupils should be stressed.

Dr. David Cogan, Cambridge, Mass: Six patients with Adie's syndrome whose sensitivity to physostigmine salicylate I have tested might be worth mentioning. I used minimal concentrations of the drug ($\frac{1}{128}$ per cent). All the patients had uniocular involvement, so the reaction of the eye with the tonic pupil could be compared with the other, presumably normal eye. It was my impression that the two eyes reacted equally; that is, the induced miosis came on at about the same time, was equally marked and lasted for approximately the same period in the two eyes. I should be interested to hear from Dr. Scheie and Dr. Adler whether or not they have had any experience with physostigmine salicylate. It seems to me that the significance of the reaction to this drug is: In the presence of a postganglionic lesion the reaction to the drug, unlike that to acetylbetamethylcholine, should be less on the abnormal side than on the normal.

Another question I should like to ask is this: If the lesion is simply an interruption of the pathway of the peripheral nerves to the pupil, would one not expect tonic pupils with palsy of the peripheral nerves that causes internal ophthalmoplegia?

Dr. Harold G. Scheie, Philadelphia: In regard to Dr. Cogan's remarks, we did use physostigmine salicylate in a very dilute solution, as dilute as $\frac{1}{256}$ per cent, and constriction occurred in the tonic pupils whereas it did not occur in the normal pupil. We cannot explain the differences in our findings, but we made control tests with the drug on normal subjects, I think 30, and it was not until approximately a dilution of $\frac{1}{64}$ to $\frac{1}{32}$ per cent was reached that the normal pupil was affected; even when we used a 0.25 per cent solution the tonic pupil decreased in size much quicker than the normal pupil. The amount of miosis was about the same.

As to the question about internal ophthalmoplegia, I think that until one knows whether the lesion is peripheral or central nothing can be said. If the peripheral nerves are damaged in glaucoma, why should tonic pupils not develop? I have not seen any.

I should like to conclude by emphasizing what Dr. Adler has said. We have presented some experimental evidence which points to a lesion peripheral to the ciliary ganglion but have not ruled out something

inherent in the muscle itself. The problem might be approached as has Thompson's disease. Harvey has written a paper in which he has shown by the use of quinine that the disturbance in myotonic goats is one of the synapse. We are working on some other theories in an attempt to localize the site of the disturbance in tonic pupils.

Protein Content of the Reformed Aqueous Humor in Man. Dr. Peter C. Kronfeld, Chicago, and Drs. C. K. Lin and T. H. Lou, Peiping, China.

The protein content of the reformed aqueous was determined in patients with apparently normal eyes, in patients with chronic retrobulbar neuritis and in patients with atrophy of the optic nerve. Repeated determinations in the same patient revealed slight variations in the same eye, slightly greater variations in both eyes of the same patient and large variations in different patients. The portion of the chamber contents which was aspirated and the local anesthetic used before the withdrawal of the aqueous were found to have a marked influence on the protein content, whereas the time interval between the first and the second puncture—within the limits of from ten to sixty minutes—had no noticeable effect. In order to account for the marked variations of protein content of the reformed aqueous in different persons, a strong individual factor must be assumed, but its nature has so far not been determined.

DISCUSSION

Dr. J. S. Friedenwald, Baltimore: I have been much interested in the series of studies which Dr. Kronfeld has conducted for a number of years on the reformation of aqueous after puncture of the anterior chamber. This has been a daring undertaking on his part and has, I think, yielded information of material value. In the present instance the aspect that seems to me of greatest significance and that which Dr. Kronfeld has emphasized most strongly is the variation in protein concentration of the reformed aqueous which appears to be independent, to some degree at least, of the amount of fluid being formed. This fact seems to me to be one of the most important keys to the understanding of the mechanism of the formation of intraocular fluid and the control of intraocular pressure. During the last few years there has been more and more evidence indicating that the whole intraocular vascular bed cannot be considered as a functional unit, that there are independent portions of the vascular bed within the eye and that these independent portions, namely, the vessels of the ciliary body, the iris and the vascular connections of Schlemm's canal, react separately and more or less independently to those factors which disturb the intraocular vascular situation. The effect on intraocular pressure of vasomotor reactions on these different vascular beds is different, and the variations in the reactions of these different beds account for much of the complexity of the phenomena of intraocular fluid balance. In some experiments which I reported to this society last year I was able to show that the reformation of intraocular fluid could be almost completely inhibited in guinea pigs by depriving them of vitamin C, and I gave reasons for believing

that vitamin C was a necessary component for the mechanism of secretion of intraocular fluid in the ciliary body. There is no reason to believe that the deprivation of vitamin C should decrease capillary permeability. On the contrary, one would expect any influence in relation to capillary permeability to be in the opposite direction. I concluded from these experiments, therefore, that most of the water which enters the eye after puncture of the anterior chamber comes via the vascular system of the ciliary body and that the iris contributes only a relatively small fraction to the water transfer. The experiments of Dr. Kronfeld and his associates suggest that a large part of the protein of the second aqueous comes from the iris vessels. It is possible that variations in the relative contributions of these two vascular beds might explain the paradox that he has uncovered.

DR. PETER C. KRONFELD, Chicago: On the spur of the moment, I cannot say anything further in regard to the possibilities pointed out by Dr. Friedenwald.

Experimental Iritis in Rabbits Produced by Coliform Bacilli Isolated from the Upper Respiratory Tract of Man. DR. CONRAD BERENS and MISS EDITH L. NILSON, New York.

Cultures of material from the upper respiratory tracts of 216 patients with inflammatory ocular diseases showed the presence of gram-negative bacilli, such as Bacillus coli, Aerobacter aerogenes or Friedländer's bacillus, and various non-lactose-fermenters in 30 per cent of cases. Brain-heart infusion cultures of the majority of these coliform bacilli when injected intravenously into rabbits produced an acute transitory iritis. A comparison of the effects of respiratory tract and miscellaneous fecal coliform strains showed the average degree of iritis produced by the respiratory tract strains to be much greater (mortality 20 per cent) than that produced by the fecal strains (no deaths). The amount of culture of an iritis-producing coliform bacillus required to produce maximum symptoms was approximately one fifteenth of that of an iritis-producing streptococcus necessary to produce the same results. Seitz filtrates prepared from cultures of iritis-producing coliform bacilli obtained from the upper respiratory tract produced the same intensity of iritis as the cultures from which the filtrates were prepared when similar amounts of each were injected intravenously into rabbits. Both cultures and filtrates lost their iritis-producing and lethal properties with age, though the rate of diminution varied in different strains. It was possible to produce a temporary immunity to these organisms by giving the rabbits one or more intravenous injections of small doses of culture or filtrate.

DISCUSSION

DR. H. M. LANGDON, Philadelphia: I should like to ask Dr. Berens whether the inflammatory conditions in the eye were limited entirely to the iris or whether they involved the deeper structures and also whether any other structures of the body were involved in the inflammatory results.

DR. CONRAD BERENS, New York: There seemed to be little involvement of the other structures of the eye. In some cases we saw some

involvement of the choroid, but the main lesion was acute iridocyclitis. This led to the publication of a paper (*J. Exper. Med.* **71:** 129 [Feb.] 1940) from the department of pathology of Cornell University Medical College on the relation of the structure of the villi of the joints to the ciliary processes, because one of our primary interests has been the relation of chronic arthritis to inflammatory ocular lesions.

As far as the question of the other organs of the body being involved, they may be somewhat affected by the inflammatory process, as one would expect with such a highly toxic organism.

Function and Structure in the Eye. Dr. Edward Jackson, Denver.

The dioptric media of the eye and the membranes that bound them have transparency, a property possessed by no other tissue of the body. Their curved surfaces, essential to the optical function of the eye, are preserved and recovered with wonderful accuracy. These adaptations have not been explained by histology or biochemistry. They may be better understood from conditions encountered in clinical experience, e. g., from the processes of recovery from injury. The part played in metabolism by the membranes of Bowman and Descemet, by the lens capsule and lens fibers and by the capillary endothelium is to be considered. The resilience of the optical apparatus of the eye is equally important and mysterious. This is possessed by other tissues but not in such high degree. The surface curvatures and their permanence and adaptability are as essential to vision as the transparency of the optical parts. The development and maintenance of this living optical instrument must be studied from all points of view, and clinical observations will help toward a better understanding.

Histologic Study of an Eye from a Child with Arachnodactyly. Dr. Georgiana Dvorak-Theobald, Oak Park, Ill.

The measurements of this eye were given at the seventy-second annual meeting of the society in the discussion of Dr. Lloyd's paper, "A Second Group of Arachnodactyly Cases," and are printed in the transactions (**34:** 47, 1936). There is an incomplete separation between the iris and the trabeculae, especially on the nasal side, where the trabeculae and the canal of Schlemm are not so well defined.

The iris shows only an occasional rudimentary crypt of Fuchs. The sphincter of the pupil is well developed and is about 2 mm. in width (fourth fetal month). The dilator of the pupil is not developed (it develops after the sixth fetal month). The posterior layer of the pars iridica retinae is pigmented only over the pupillary half of the iris, and from the unpigmented cells occasional fibers extend toward the ciliary processes. The deposit of pigment in this posterior layer of ectodermal iris occurs, according to Ida Mann, from the third to the seventh month.

The histologic appearance of the other tissues resembles that found in eyes that are myopic from other causes.

The myopia occurred before bony structures were formed. From the histologic picture, the abnormality and arrested development must have occurred between the eighth week and the sixth month of fetal life.

Significance of Sturm's Interval in Refraction. Dr. A. D. Prangen, Rochester, Minn.

The physical phenomena incident to the conoid of Sturm and Sturm's astigmatic interval have direct bearing and application in clinical refraction. By keeping in mind a clear picture of the phenomena of Sturm, the examiner is able to visualize and interpret more readily the phenomena of astigmatism, both subjective and objective. In correcting astigmatism, an attempt is made to obliterate Sturm's astigmatic interval, either partially or completely. The character and behavior of the varying images of Sturm's interval are important to keep in mind while attempting to correct and to prescribe for astigmatism. Frequently it is advisable to correct astigmatism partially. The phenomena of Sturm help the examiner to understand this necessity and to guide him in writing a better type of prescription, using equivalent, balanced reduction of cylinder with change of sphere. Such equivalent formulas are discussed. A few patients who have astigmatism are more comfortable without astigmatic correction, and this is explained optically by Sturm's phenomena.

DISCUSSION

Dr. W. B. Lancaster, Boston: I wish to add a word of approval for Dr. Prangen's insistence on the importance of the conoid of Sturm. The experience of the American Board of Ophthalmology shows that the candidates who come up for examination do not understand astigmatism. For example, they do not understand the use of astigmatic charts, and naturally that is because they are not taught. Therefore, I am addressing myself especially to the teachers, since most of them, or at least the heads of departments, are members of this society.

I should like to ask Dr. Prangen and others who discuss this paper what they would say to the student who brings Gullstrand's statement to their attention. If they read Helmholtz' "Physiological Optics," and every student should be required to read it (some of the mathematics may be over his head, but it is not all mathematics), they will discover that Gullstrand stated: "The path of the rays in the human eye does not even approximate the conoid of Sturm." Sturm was a mathematician, and comparison of his theories with those of Gullstrand is like comparing high school geometry to postgraduate university mathematics, and even Gullstrand when he tried to reduce the refraction of the eye to mathematical formulas found that he had to give up the complete solution and depend on actual observation, which he called stigmatoscopy, i. e., study of the image subjectively as it appears on the retina when the focal place is moved forward and back.

What is the reason for Gullstrand's statement that the astigmatic eye does not correspond to the conoid of Sturm? The reason is that the eye has so many aberrations. Here is the simplest aberration of the spherical lens. The axial rays, those adjacent to the axis of the lens, come to a focus here (illustrating) and the marginal rays come to a focus at a distinctly different point nearer the lens. Sturm took no account of that. The formulas of Sturm and of most of the writers on theoretic optics are valid for an infinitely small area at the very center of the lens. They take no account of the peripheral rays. The human eye, of course, has to take care of the rays in the whole area of

the pupil. Hence students should be taught the practical application of spherical aberration, especially with dilated pupils.

In this diagram is shown the same type of astigmatism as is shown in the glass lens. Some of the rays—the peripheral rays—come to focus here. The central rays come to a focus here at a point farther from the lens. This diagram, by the way, is drawn from Tscherning's own eye (Tscherning, M. H. E.: Physiologic Optics, Philadelphia, Keystone Publishing Co., 1924, p. 146). In another meridian, the vertical, there is an asymmetric condition. The rays are much more strongly refracted by the lower half of the cornea than they are from the upper half. By studying the different parts of his cornea with the pupil dilated, Tscherning found at the center of the cornea myopia of 1 D.; just above the center, emmetropia; near the lower margin, myopia of 3.5 D.; near the nasal margin, myopia of 2 D.; at the temporal margin, myopia of 2.75 D., and near the superior margin, hypermetropia of 1 D. The refraction of the rays by different parts of the cornea and lens is not as simple as the conoid of Sturm by any means.

I asked myself what the answer to my question would be. I ought to give my own answer, which is that the lenses which are used to correct errors of refraction, spheres and cylinders, refract light precisely according to the conoid of Sturm, and therefore the best one can do in correcting errors of refraction is to use these simple regular lenses and select that combination which corrects the largest possible part of the mixed errors of the eye. It would be a great help to students to make these experiments on each other and on themselves and to study the effect of the different lenses on the image.

I should like to show the effect of acccomodation on the conoid of Sturm as demonstrated by the projection lens of the lantern. By slipping this pasteboard attachment over the projection lens, I can insert a cylindric lens from the trial case before the projection lens, thus making the projection system astigmatic. When a slide is inserted which consists of an opaque card with a tiny hole in the center, the image thrown on the screen is a small spot of light, virtually a point. A —0.25 cylinder changes the image of the point to a line. Next, instead of a point, a slide with two narrow slits (lines) at right angles to each other is inserted, first without the cylinder. A sharply defined image of the lines is thrown on the screen. Every point of each line is imaged as a point on the screen, and so the lines are sharply focused. A —0.25 cylinder is now placed in front of the projection lens with its axis parallel to one of the lines. The image of every point is now a line. The result is: One line remains sharp on the screen because every point in it is a short narrow line parallel to the line itself; the other line is very blurred because every point of it is imaged as a line at right angles to the line itself. What I wish to demonstrate is the effect of accommodation on this image of the cross consisting of one sharp line and a very blurred line at right angles to it. As the lantern operator focuses the instrument in the usual way, a position is found where both lines are alike. There is no visible astigmatic effect. Of course, the lantern lens system can change its focus only as a whole, not more in one meridian than in another; in other words, there is no possibility of producing what is called in the eye "astigmatic accommodation"—greater action of the ciliary muscle in one meridian than in another, thus

compensating or correcting astigmatism by an unequal contraction of the ciliary muscle. The neutralization of astigmatism must be accomplished in some other way. Obviously, it is by so focusing the lantern that the image is at the interfocal circle of the conoid of Sturm; then the image of every point is a small circle and the lines on the screen are both alike but slightly blurred.

Finally, a slide is inserted showing an astigmatic dial, first without the —0.25 cylinder. All the lines are clear and equal. Next, the —0.25 cylinder is used. A small group of lines is clear and the others are blurred. If the cylinder is rotated, the group of clear lines is rotated, showing how easy it is to locate the axis of the cylinder. If a + 0.25 cylinder is inserted with its axis parallel to the —0.25 cylinder, all the lines will be alike again. Without adding a plus cylinder to neutralize or correct the —0.25 cylinder but merely by focusing (accommodating), all the lines are made absolutely equal but slightly blurred.

The importance of this clinically is great. It has never been demonstrated that unequal accommodation is a function of the normal eye. The normal eye neutralizes astigmatism by normal accommodation. This is best prevented by fogging, since this avoids the confusing and misleading aberrations introduced when the pupil is widely dilated. It is to be regretted that this art is not more widely taught.

Dr. Alfred Cowan, Philadelphia: In teaching optics at the University of Pennsylvania the instructors always endeavor to show how the theory of optics may be applied to everyday practical work. The students do the experiments themselves and having actually set up an optical system on a bench are able to visualize Sturm's conoid and to know exactly what it means. They must realize that Sturm's conoid is a figure composed of a series of diffusion images of an object point as seen by a spherocylindric optical system. In such a figure as Sturm's conoid the first focal line by the refraction of one meridian of this system and the second focal line at the focal distance of the other meridian are at right angles to each other. Between these two lines is what is commonly known as the circle of least confusion. The distance between the two focal lines indicates the amount of astigmatism of the two systems. If they coincide, there will be no astigmatism; therefore, the greater the distance between the two focal lines, the greater the amount of astigmatism. The circle of least confusion is not a circle except when the aperture is a circle, so that the term "circle of least confusion" applies only to a system which has a circular aperture. If the aperture were square, the diffusion image at this place would be square. However, in every system of this kind every image is a diffusion image. The image of an object point is never a point at any place in the system. In applying this to the eye the retina may be placed anywhere, the type of astigmatism depending on where it is placed. If it is at the first focal line there is simple hypermetropic astigmatism; if behind the second focal line, myopic astigmatism; in front of the first focal line, compound hypermetropic astigmatism, and if between the two focal lines, mixed astigmatism.

What is accomplished with lenses? The whole figure may be shifted with spherical lenses, bringing it forward or backward. When spherical lenses are used the distance between the two focal lines which indicates

the amount of astigmatism is never affected. Only a cylindric lens can approximate or farther separate those two lines.

What Dr. Prangen has said about equivalent lenses is important to a certain extent, but only quantitatively, not qualitatively. Quantitatively, one may assume roughly that the amount of curvature in a 2 D. cyl. approximates the total amount of curvature of a 1 D. sph., but qualitatively there is no such equivalence. I think that the first ophthalmologist who used these equivalents was Dr. Jackson, as recorded in a paper published some time ago. For his purpose it was fine. A crossed cylinder furnishes a good example of what I mean. A + 0.50 D. cyl. crossed with a — 0.50 D. cyl. is nothing but a spherocylinder with a +0.50 D. sph. and a —1.0 D. cyl. or a —0.50 D. sph. and a + 1.0 D. cyl. If a 0.50 D. sph. of one denomination is plotted against a 1.0 D. cyl. of the opposite denomination (using the equivalent table) it is seen that a 0.50 D. sph. would neutralize exactly 1.0 D. cyl. of the other denomination. This is an example of a lens from the equivalent standpoint which is neutral or would be called plano from the quantitative standpoint, but qualitatively it is still a 1.0 D. cyl. This is one of the reasons for using the crossed cylinder. When the patient chooses a point in Sturm's conoid by accommodation or otherwise, he chooses a place where there is the least amount of astigmatic distortion. At best, he still has a diffusion image of the type which is found with a spherical error. His visual acuity should not be better than would be indicated by the amount of spherical error from the first or second focal line to the circle of least confusion.

It is true that Gullstrand has shown actually and mathematically that there is no such thing as this figure, but after he showed conclusively that he was right, he seemed to forget and used a figure such as this, which he called the fiction of rays, to demonstrate everything he spoke of after that. The eye is a different type of optical instrument than the manufactured ones. It is a dynamic instrument which can change its focal power and pick a place for itself. In the eye, as Gullstrand stated, there is no fiction of rays as Sturm's conoid but a small amount of aberration that would make the lines form a figure which is so complicated that it can hardly be drawn on a plane surface; but the eye chooses the proper place by its ability to accommodate, and while the eye may receive an image which has a wide area of aberration around it, by its ability to ignore the peripheral portion of the image and to pick out and interpret the image it is still able to see distinctly, even with the aberration which Gullstrand has shown.

DR. F. H. VERHOEFF, Boston: I shall try to present this situation from a different point of view. I would say that the conception of Sturm's interval is an important one and that every one who undertakes refraction should pass through the stage of this conception. It represents an embryonic stage in the development of knowledge of refraction of the eye, and some persons never get beyond it. After the conception is thoroughly understood, one has to realize to what extent it cannot be applied. To understand certain phenomena, the conception must be modified. To explain certain things, aberrations must be considered in addition. For example, simple spherical aberration combined with astigmatism, when considered in the light of Sturm's interval,

will explain many cases of monocular diplopia. Similarly, consideration of aberrations in addition to Sturm's interval is necessary to explain the cases of so-called aphakic accommodation. If one goes still further, one has to consider also chromatic aberrations, diffraction and even polarization to explain certain phenomena. The same problem is encountered in every field. One tries to simplify everything by making a dogmatic assumption which conveys a certain conception, and then one spends the rest of a lifetime saying that the conception is not exactly a true one. But the fact that it is not exactly true does not mean that the conception is not a valuable one.

DR. EDWARD JACKSON, Denver: This has been an interesting and instructive discussion of optical theory, but perhaps it has not been carried into the realm of ordinary practice. After Sturm and Helmholtz had written, skiascopy was introduced as a means of measuring refraction of the eye. With skiascopic study of the dilated pupil, the outstanding fact that impresses one is that the refraction is unequal in different parts of the pupil. As a rule the refraction is greater in the margin of the pupil (there is less hyperopia and more myopia), for the rays that come through the periphery of the pupil than for those which come through the center. Also, astigmatism varies in different parts of the pupil, in meridians and in amount. The so-called linear shadows are not lines; they are rather areas of light and shadow that move in different directions in different parts of the pupil. These aberrations disappear when the pupil is contracted. For this reason the final determination of subjective refraction of eyes should be made in a relatively strong light. The ciliary muscle may or may not be completely relaxed. The habits of the patient are important. I find the most useful rule is never to prescribe a spherocylindric correction on one determination, but at another time and on another day, when the patient has rested the eyes as much as possible, to make a second determination, starting with what I found before and correcting the mistakes of my first examination. Sometimes I have to see a pair of eyes five times before I can satisfy myself that I have the correction for the contracted pupils that will be of the greatest service to the patient.

The obliquity of the glass makes a difference. Sometimes when a patient asks what astigmatism is, I take a spherical lens from the trial case and hold it up, perhaps with the sun shining in the window, and get a small circular image of the sun; I then turn that spherical lens a little obliquely and immediately introduce the interval of Sturm. At one distance one gets a vertical line and at another distance a horizontal line. This is what happens when a patient looks through different parts of his glasses; the larger the glasses, the more difficulty he will have. The patient looking obliquely through his glasses will get a different interval of Sturm from the one he will get when he is looking exactly perpendicular. All these things have to be considered. One practical idea I find most effective and which I prescribe for college and high school students at the end of the semester, when they are studying for examinations, is that if they have to have a pair of new glasses, they can get more out of an hour's reading by not looking at the page all the time, but for short intervals, perhaps every minute, closing the eyes and

thinking about what they have just read and letting the eyes rest. Every time the eyes rest, the image is most distinct when they are first opened. At such times one catches the relaxed eye—the eye that theoretically has been corrected with the lens. The students quickly overcome the difficulties that Dr. Prangen has so graphically depicted and that are so widely distributed in different parts of vertical objects. Thus, they quickly learn how to see through their glasses, for every one who gets a new correction has to learn this to some extent.

DR. H. M. LANGDON, Philadelphia: To all who are practicing ophthalmology, this is an important subject. It is bound to be, because 75 per cent of the ophthalmologist's work is of this type. There are two points I should like to speak of especially. The first concerns Dr. Lancaster's suggestion that knowledge of the astigmatic dial for the study of the effect of Sturm's conoid will help students in refraction. I agree with him and think the astigmatic dial is useful for students of refraction, but I do not think the ordinary astigmatic dials are of great service. To get the greatest benefit of the principle, one should use the dial made up by Herbert Ives, but not with the two gratings. Take one grating out, and use merely the lines.

The second point I wish to bring up is something which has not been mentioned here, and that is the comfort of the patient. In talking to students I tell them they have not done a successful refraction unless they achieve good distance vision and a good near point and bring about relief of symptoms. Next is the question of partial correction of astigmatism. This depends, I think, entirely on the conditions that Dr. Prangen has described. There will be only a small fraction of 1 per cent of persons who will not take the correction if the error is measured accurately and due allowance is made for the play of the ciliary muscle afterward for changes in the spherical lens. I do not believe in changing the cylinder. I have seen persons with 1.5 D. of astigmatism with normal vision and a good near point and no symptoms. There is no reason for urging such persons to wear a correction if they are comfortable without it. That is a rather extreme example, but I have seen such persons. Therefore, the correction of astigmatism depends on the subjective symptoms of the patient, and when one gets into this subject one is getting into a wide field.

Secondary Glaucoma Due to the Lens. DR. PARKER HEATH, Detroit.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

A Rare Form of Glaucoma. DR. S. JUDD BEACH and DR. E. E. HOLT, Portland, Me.

It has long been regarded as axiomatic that the use of drugs to dilate the pupil is contraindicated in persons predisposed to glaucoma. Recently the contrary view has been advocated, that precipitation of an acute attack in these cases is not wholly undesirable. This is advocated, of course, on the ground that earlier recognition of the disease leads to more prompt and effective treatment. Thus so-called provocative tests have been widely used in doubtful cases. Some writers even

appear rather to minimize the danger from accidental increase of tension during examination of refraction.

This report points out data apparently overlooked in recent reviews of the literature and also presents a number of hitherto unpublished cases.

DISCUSSION

DR. JOHN GREEN, St. Louis: The case which I have to report is not a case of increase in tension following cycloplegia preparatory to refraction. Unilateral acute glaucoma developed in an elderly woman, and for one week she was enthusiastically treated with atropine by her physician. By the time I saw her she had very high tension and perception of light. Prompt basal iridectomy reduced the intraocular pressure, but there was no restoration of vision. Miotics were prescribed for the other eye, although it had normal tension, a full field and fairly good vision. There were, however, some changes in the central portion of the lens.

A few weeks later the patient returned, and in my zeal to find out more about the intraocular pressure and the periphery of the lens, I injudiciously, as it turned out, used a drop of a 1 per cent solution of epinephrine bitartrate in the unaffected eye. Within an hour the pupil had dilated, and the woman was suffering great pain. The tension went up to 50 mm. of mercury (Schiötz), with a concomitant decrease in vision. I put the patient in the hospital and spent the entire night ineffectively treating her with pilocarpine, then physostigmine salicylate and finally aminoglaucosan (a 10 per cent solution of histamine hydrochloride). The following morning I performed a basal iridectomy, which effected a prompt and permanent reduction in tension.

I learned from a colleague, whom she later consulted, that despite my efforts before, during and after operation she did not think at all kindly of me, although final vision with correction was 20/40 +.

When I proposed the use of a solution of epinephrine bitartrate (1 or 2 per cent), I suggested that it be used cautiously and only in cases of chronic simple glaucoma and as an adjuvant to miotics. That warning did not deter some ophthalmologists from using it in cases of acute glaucoma, with disastrous results.

DR. ALLEN GREENWOOD, Boston: Some thirty years ago I had 2 patients (women) in whom acute glaucoma developed following the use of a mydriatic. The attack of the first patient came on in the afternoon, and I was called to Cambridge from my office to see her. The other patient lived about 50 miles away from the office, and I was called at 10 o'clock at night to treat her. Both patients recovered without trouble.

DR. HANS BARKAN, San Francisco: May I ask Dr. Beach whether there are any records available regarding the time of the month in which these women had glaucoma? It is conceivable that when one dilates the pupil, especially in younger women who have not reached the age of the menopause, one may be doing it in a certain number of them during their menstrual period? Has it ever been ascertained whether this is a fact, and if so, would it have a bearing on whether this form of glaucoma in women of that age is a form of vasomotor instability and that in dilating the pupils one has added a certain amount of local insult? That may explain why the glaucoma disappears spontaneously or why it

does not tend to become permanent. I should like to report 1 case of this occurrence in the last few months which may throw some light on the matter. The patient was a woman 48 years of age. During her menstrual period she came for refraction, and as I had reason to believe that she might have hyperphoria I put on a patch. Twenty-four hours later, I took off the patch and found no phoria. I was on the point of saying "That is all for today," when I noticed that the cornea in that eye was dull and cloudy. The patient had had no pain, and in my consternation I said nothing but that I should like to have her stay while I put in a few drops of pilocarpine. The tension of 70 (McLean) dropped to normal within two or three days, and she has had no trouble since. This was a case of angiovascular instability precipitated by the simple process of dilating the pupil for twenty-four hours under a patch. I should like to ask if Dr. Beach has any records or whether he knows of any regarding coincidence of menstrual period and development of glaucoma after the use of a mydriatic.

DR. E. E. HOLT JR., Portland, Me.: In reply to Dr. Barkan's question, I should say that Dr. Beach has no record of this occurrence during the menstrual period.

This paper stresses the possibility of increased intraocular pressure after the use of a cycloplegic and indicates that the frequency of its occurrence is more than 1 in 10,000 cases. However, such episodes should tend to make one a little more cautious but should not deter one from using these drugs. In over half the cases the anterior chamber was shallow and the pupils were somewhat sluggish. These signs may be unfavorable although they are not positive. In the case of increased intraocular pressure it seems reasonable to deduce that control may be facilitated if the methods of Beach and McAdams as presented before this society in 1937 are used. As to the use of miotics after a cycloplegic, I have followed the suggestions of H. Gifford as given in his paper in 1916. Physostigmine salicylate (0.16 to 0.25 per cent) is instilled two or three times after the examination is concluded, and the patient is observed for an hour at least; if the patient lives out of town, a weak solution of mio.ic is prescribed two or three times a day for three or four days.

Intra Diem Tension Studies in Chronic Simple Glaucoma. DR. PAUL A. CHANDLER, Boston.

This article was published in full in the July issue of the ARCHIVES, page 62.

DISCUSSION

DR. HANS BARKAN, San Francisco: Dr. Chandler's paper has covered the subject so well that I should only like to add that since having seen the first articles on the subject in *Acta ophthalmologica* about ten or twelve years ago, I have used this procedure for a number of years and find it of great benefit for certain types of patients. My practice has been to put them in bed in the hospital and to give very small doses of sodium bromide or a barbiturate in order to get as much relaxation as possible. The patients who have been willing to do this for me, with consequent benefits to themselves and myself, have been

mostly women, simply because women are able to manage a four day lay-off more easily than the average business man.

The next group I have been able to hospitalize is made up of patients from the country. To emphasize the gravity of the situation to these persons, knowing that they come to the city for just one day, and in order to make one's position with them somewhat more emphatic, it is well worth while from the practical standpoint of the relation of physician to patient to tell them you want them in the hospital for a short stay and to explain why. One should make an effort to tie this type of patient to the doctor more than other patients, especially if one can give them, as one frequently can after four days of examination, a favorable report. One can say, "You will probably not have to have an operation, at least not for a number of years," or, "You will probably not lose any more vision if you continue these treatments as systematically as you have in the hospital, but you must do so and you must return in three or four months in order to have this painless and not particularly expensive test (because it is not much more expensive in the hospital than in a hotel) repeated." If on repetition it is shown that the tension is low, one knows that one is safely on the way. I should like to emphasize that this test is very valuable to use for selected patients.

Dr. Peter C. Kronfeld, Chicago: There is considerable disagreement in the literature with regard to the classification of cases of Dr. Chandler's group 1. While some ophthalmologists consider them to be cases of chronic compensated glaucoma during a phase of low tension or remission, others (Gradle) call them cases of glaucoma with an unusually low tension tolerance. A third group of authors has labeled cases of this type pseudoglaucoma, the term implying that the mechanism of the visual loss in these cases is not the glaucomatous one. A vascular disease of the optic nerve is usually assumed to be the cause of the atrophy of the optic nerve with glaucomatous excavation which is present in these cases.

Dr. Chandler has performed trephine operations in cases of this type and reports no further loss of visual function as long as the eyes remained hypotonic. This observation, in my opinion, constitutes a strong argument in favor of the glaucomatous nature of the disease in these cases, since a vascular disease of the retrobulbar or intracranial portion of the optic nerve could hardly be expected to be affected by a state of ocular hypotony.

Dr. S. J. Beach, Portland, Me.: When the fields are characteristic, McAdams and I believe these cases to be cases of low tension glaucoma. When the condition does not respond to miotics, it may often be arrested by trephination.

Dr. Paul Chandler, Boston: For my part, the experience with the few cases in which I have operated has convinced me that these are instances of true glaucoma, particularly case 1. In this case there was definite loss of field although the patient used a miotic faithfully; operation was performed, and during the next four years there was not the slightest loss of field and tension was maintained at around 10 or 12 mm. of mercury; then again a loss of field occurred and the tension was found to be 20 on account of partial closure of the filtering scar.

Paralysis of Both External and Internal Rectus Muscles with Preservation of Convergence in a Case of Intramedullary Tumor of the Pons. Dr. Warren S. Reese and Dr. Joseph C. Yaskin, Philadelphia.

A case of intramedullary tumor of the pons with extension into the fourth ventricle is reported. The earliest objective abnormalities included bilateral paralysis of the sixth nerve and paralysis of both internal rectus muscles for lateral movement but not for convergence. Later well marked signs of a tumor of the posterior fossa developed, and at operation an astrocytoma was found protruding into the fourth ventricle. The possible reasons for the preservation of convergence are discussed.

DISCUSSION

Dr. Francis Heed Adler, Philadelphia: The value of this interesting case lies in the ability of localizing the lesion that can produce these symptoms. The symptoms were first pointed out by L'hermitte about 1921 as indicating that the lesion must be in the posterior longitudinal bundle, somewhere between the nucleus of the sixth nerve and that of the third nerve; he called the condition "ophthalmoplegia internuclearis." He also pointed out on the basis of a number of cases that one can have two forms of the disease. As one can readily appreciate, the fibers coming from the pontile center, which lies close to the nucleus of the sixth nerve, deviate. One set goes to the nucleus of the third nerve and the other set to the nucleus of the sixth nerve. The pathway of fibers from the pontile center to the nucleus of the sixth nerve, which is closely adjacent to it, is very short, and those fibers going to the nucleus of the third nerve must pursue a longer course and separate from the former. Accordingly, one can have two forms of the disease, ophthalmoplegia internuclearis anterior in which the lateral rectus muscles are spared and the internal rectus muscles are only affected in conjugate lateral movements, and the second form, the posterior form, in which the lateral rectus muscles alone are involved. This form is more difficult to diagnose.

Those here will remember a case of the simple form of ophthalmoplegia internuclearis anterior reported by Bielschowsky. The patient was unable on looking to the right to move the left eye to the right. When he looked to the left the right eye failed to move to the left, but on convergence both internal rectus muscles were shown to be intact. This was thought to be due to a small hemorrhage in the posterior longitudinal bundle. The patient subsequently recovered. I have seen a similar case in which the condition was the result of herpetic encephalitis following herpes zoster. The patient never recovered. The case described by Dr. Reese and Dr. Yaskin is, I believe, exactly similar to this one, with the exception that the lesion has extended farther backward and now has affected the nucleus of both sixth nerves; this is proved by the fact that the eyes fail to respond to vestibular stimulation. The great value of the case lies in the fact that one can localize the lesion well within 2 cm., and the practical point is that the lesion in such cases is rarely amenable to surgical intervention.

Dr. Warren S. Reese, Philadelphia: If I understood Dr. Adler to say that he felt that in this case the nuclei of the abducens nerve

are involved, I disagree with him. I think that the centers for lateral movements, which are close to these nuclei, are involved, but I do not think that the nuclei themselves are affected.

Late Developments in a Case of Gyrate Atrophy of the Choroid and Retina (Fuchs). Dr. Hunter H. McGuire and Dr. W. P. McGuire, Winchester, Va.

The present communication is concerned with a description of the advanced stages of a case of gyrate atrophy of the choroid and retina, originally presented at the 1932 meeting of the section on Ophthalmology of the American Medical Association. This condition was first described by Fuchs at the Heidelberg Congress in 1895. Drawings of the fundi and visual fields of the early and late developments are presented, together with an abstract of all the available literature.

DISCUSSION

Dr. John Green, St. Louis: I should like to ask Dr. McGuire whether general therapy had any effect on this process.

Dr. W. P. McGuire, Winchester, Va.: No therapy that we have tried or that has been tried by other physicians has had any effect. The last time we saw the patient in October we put him on a diet very high in vitamins, and we have not seen him since. Whether it had any effect, I do not know. I was rather doubtful that it would have.

Heparin in the Treatment of Complete Thrombosis of the Central Retinal Vein. Dr. Alexander E. MacDonald, Toronto, Canada.

Following the discovery of heparin by Howell in 1916 and its purification by Best and his associates in 1933, Murray, in the Hunterian Lecture of 1939, reported its successful intravenous use in many surgical conditions. The clotting of blood is not a reversible process, and, theoretically, there would seem to be little justification for the use of heparin after complete thrombosis of the central retinal vein. In practice, however, Continental observers have reported recovery of normal vision as a result of the intravenous use of the drug.

A review of the literature and a report of a patient treated with heparin for complete thrombosis of the central retinal vein are presented.

DISCUSSION

Dr. Parker Heath, Detroit: I should like to add 1 case to those reported, that of a girl 18 years old. She had what appeared to be central retinal occlusion—clinically called arterial thrombosis—and there were some hemorrhages. She was given heparin for five days by the continuous drip method. Some intermissions in treatment were necessary because she was disturbed; she had fainting spells, a thin, rapid pulse and slight rise in temperature of less than 1 degree. She did not improve. The condition in the fundus progressed to atrophy of the nerve head and questionable perception of light in the eye.

Dr. Sanford R. Gifford, Chicago: I should like to place on record 2 cases in which heparin was given rather late, in one, a month after

the thrombosis occurred, and in another in two weeks, with no improvement in vision. One man was given about $60 worth of heparin, so I am glad the price has gone down. I think it should be tried in cases of earlier involvement.

Dr. F. H. Verhoeff, Boston: It always annoys me to hear thrombosis of the central retinal vein spoken of as if such a diagnosis could be made ophthalmoscopically. In all the textbooks, pictures showing the effects of obstruction of the central retinal vein are labeled "thrombosis of the central retinal vein." It is probable that only 1 in 100 such cases is there really thrombosis of this vein. All that is known from the ophthalmoscopic picture is that there are obstructions in serial cross sections. I have never been lucky enough to find one eye in which there was thrombosis of the central retinal vein. There has been endophlebitis and not the slightest evidence of thrombosis, even as a terminal event. In these cases it would be ridiculous to use heparin, because even if one got rid of the supposed thrombosis, one would have the vein blocked by tissue on which the heparin could have no effect. I think actual thrombosis of the central retinal vein occasionally occurs in young persons. In some of these cases there is a history of influenza or phlebitis or some other infection to account for the thrombosis. These are exceptional cases, and the remarkable thing is that in these cases complete recovery of vision is apt to occur without the use of heparin or any other treatment. In an adult, thrombosis of the central vein occurs so rarely that such a diagnosis is almost never justifiable. Of course, if one wants to waste some heparin in a case of senile endophlebitis of the central retinal vein it may not do any harm. I am not so sure as to its harmlessness, however, that I would be willing to use it in any case of obstruction of the central retinal vein in an adult.

Dr. Ralph O. Rychener, Memphis, Tenn.: It is fitting that the first allusion before this society to the use of heparin in the treatment of a patient with ocular disease should come from an ophthalmologist from Toronto, where a stable, nontoxic solution of this drug suitable for clinical use has been prepared in the Connaught Laboratories by Best, Charles and Scott and where an extensive clinical investigation has been carried out on more than 400 general and surgical patients.

My interest in the possibilities of the clinical application of heparin to the treatment of thrombosis of the central vein of the retina was initiated by isolated reports of its successful employment in such cases. In December 1938 the necessity arose for offering some advice during a consultation over a male patient, aged 52, who presented such a complication and who was being prepared for an intracranial operation because of misinterpretation of the ocular findings by his referring physician. This patient was given the German product derived from dog livers; it was used somewhat cautiously in intermittent doses of low potency because of lack of familiarity with the clinical possibilities. Naturally there was no response, but the eye has retained a full form field, although the visual acuity is only 1/200. Since then Dr. E. C. Ellett and I have treated 5 other patients with partial or complete thrombosis of the central vein of the retina with the solution of heparin prepared by the Connaught Laboratories.

The first of these was the mother of my office assistant, a woman of 52, with occlusion of the superior temporal branch of the retinal vein of the left eye and reduction of visual acuity to 6/30. The Connaught Laboratories product was employed by the intermittent method of small doses at four hour intervals but without laboratory control. The response was immediate, however, and within three weeks the retinal hemorrhages had been absorbed, the vein was again normal in appearance and the visual acuity was 6/6.

Our next patient, a man of 68 with advanced general arteriosclerosis, was treated by the continuous drip method, but too cautiously, and his coagulation time was never extended beyond five and three-fourths minutes. The complete occlusion in the left eye with loss of central vision of two months' standing was unchanged by treatment. The right eye, in which there was thrombosis of the upper half of the vein which progressed to include the inferior nasal branch in the first few days of observation, so that only the inferior temporal branch remained uninvolved, retained a visual acuity of 6/10 and there was no further progression of the thrombotic process. Within three weeks after hospitalization of the patient and institution of treatment with heparin, improvement in the retinal condition was definitely distinguished by the absorption of hemorrhages and exudates. After five months there were almost no distinguishable hemorrhages in either retina and the veins were nearly normal in caliber. The visual acuity of 6/10 and ability to read Jaeger's test type 1 have been retained in the right eye. The left eye has an eccentric visual acuity of 1/120.

With this experience we gained more confidence in the reliability of the statements made about the nontoxic qualities of heparin (Connaught Laboratories) and proceeded to institute therapy to such a degree as to lengthen the coagulation time in our patients to approximately fifteen minutes.

Our next patient was a man of 69 who had lost the vision of the right eye four years previously due to trauma and secondary glaucoma, and it was enucleated because of pain. Three weeks before observation his vision had suddenly become poor, and examination disclosed complete thrombosis of the central vein in the left eye with a visual acuity of 5/60. Treatment was immediately begun, and the coagulation time was increased and maintained between sixteen and twenty minutes. Within five days he volunteered the information that his sight was improved, and this was verified two weeks later at the office, where his visual acuity was found to be 6/20 and he could read Jaeger's test type 8. This vision was maintained six months later, when the veins had become almost normal in appearance and the large retinal hemorrhages had disappeared. Minute striate hemorrhages could still be detected in association with the temporal veins and were also present about the fovea in association with a small deposit of hard exudate.

The fourth patient was a man of 67 whose total occlusion was of three weeks' duration and whose visual acuity was reduced to 1/120. His coagulation time was increased to thirteen minutes despite much difficulty in retention of the intravenous needle. No visual improvement ensued, although absorption of the retinal hemorrhages took place much

more rapidly than in any of our other patients. His convalescence was complicated by peripheral neuritis, which yielded promptly to analgesics and vitamin B therapy.

Our fifth patient, a woman of 47, should have been our best prospect for a cure, as she was observed within two days after visual failure because of venous occlusion. However, much difficulty in administering the drug was encountered due to the presence of peripheral veins of very small caliber, and her coagulation time could not be maintained at the optimum time of fifteen minutes. There was no response in improvement of visual acuity, nor was there absorption of the retinal hemorrhages in any degree similar to that observed in the previous case.

Whether heparin has any function beyond preventing an increase in the size of the thrombus, thereby allowing contraction of the clot already established and promoting parietal circulation as well as early canalization of the occluding mass, remains to be determined by further clinical investigation. Certainly the grave prognosis of thrombosis of the central vein of the retina which leads so often to secondary glaucoma and enucleation justifies this form of therapy with heparin, even though it entails hospitalization and an expense of $35 to $40 for a ten day supply of the drug. Our second, third, fourth and fifth patients were quite satisfied with the results obtained, although our expectations were not fulfilled for the complete occlusions except in the persons with only one eye. We expect to continue recommendation of this form of therapy, as during this same period we have had to remove four other eyes with complete occlusion because of secondary glaucoma from patients unable financially or otherwise to procure treatment with heparin.

In the light of past experience we will attempt treatment more vigorously in an effort to prolong the coagulation time to at least the optimum time of fifteen minutes, but we may be able to shorten the time of hospitalization and the consequent expense, which is a factor of much importance. Whether such treatment has any influence in decreasing the incidence of secondary glaucoma or recurrence must be determined by time.

I might add that in the treatment of these patients we have had none of the complications mentioned by Dr. Heath.

Dr. Paul A. Chandler, Boston: Since the problem of the treatment of thrombosis of the central retinal vein is apparently still unsettled, I should like to say one word about another possible field for the use of this drug. There are at present 2 patients in the hospital in Boston with cerebral thrombosis. One had a thrombosis of the cavernous sinus with all the typical signs. It is now twelve weeks since the onset of the patient's illness and she is about ready to go home. In the other patient thrombosis of the longitudinal sinus developed following frontal sinusitis with a subdural abscess, which was drained. This patient is now in the seventh week of his illness; the temperature is normal, and it seems probable that he will get well. When one is called in consultation to see these patients with thrombosis of the cavernous sinuses or of other large sinuses, I think one can offer a possible hope for them in the use of this drug.

Dr. A. E. MacDonald, Toronto, Canada: I wish to call the attention of those who have discussed my paper to the brilliant work on

heparin reported by Best, Scott, Murray and others, for it is outside the purpose of this paper to discuss the general considerations. In all recent experimental and clinical work heparin has been well tolerated. I should like to ask Dr. Heath if the heparin of the Connaught Laboratory was used in his case.

DR. PARKER HEATH, Detroit: Yes.

DR. A. E. MACDONALD, Toronto, Canada: I am surprised to hear this, because in 500 clinical cases there has been no death due to heparin. The second patient presented technical difficulty, as she had fine veins, and some edema of the foot resulted from the operation necessary to insert the cannula.

In regard to Dr. Verhoeff's remarks, I may say that in reviewing the literature I have been interested in the recent advances made in the knowledge of clotting, on account of the preparation of the enzymes, prothrombase, thrombase and thrombokinase in almost chemically pure state, so that the physiology of clotting is in a state of flux. I think that Dr. Verhoeff's question is best answered by referring him to the work of Belt, who made postmortem examinations, and who in 10 per cent of all cases in which autopsy was performed found definite evidence of antemortem thrombosis when the veins in all the extremities were carefully dissected and examined.

I am glad to know that Dr. Rychener has had good results. In following the progress of the disease in 1 patient, whose physician refused to let her come to the hospital early, I found an increase week by week in the area involved and in the number of hemorrhages. This was accompanied by a coincident decrease in vision. I think that this indicates a slow progression of the clotting process, which was arrested by heparin with resulting improvement in vision.

Dr. Gifford remarks that heparin is an expensive drug. This is so, as its preparation is a complicated procedure and many pounds of beef lung are required to make 1 Gm. of heparin. The cost has been gradually reduced. Heparin, I think, is as important a scientific achievement as insulin, but its application is more restricted.

Dr. Chandler will find the surgical uses of heparin discussed in Murray's 1939 Hunterian Lecture. It is a new drug, and the next decade will crystallize the knowledge of its therapeutic indications.

I may add that when heparin is used in the blood of an experimental animal it may be perfused for hours through the capsule and the scratch will show no clotting.

In closing, I want to call attention to the progressive nature of retinal thrombosis, for in a search of the literature I have not found this point mentioned; its importance can hardly be exaggerated.

Bergmeister's Papilla and Some of Its Variations. DR. RALPH I. LLOYD, Brooklyn.

The blood supply necessary for the development of the lens proceeds from a cone-shaped mass of embryonic tissue on the optic disk which branches as it passes forward to form the posterior layer of the posterior vascular sheath of the lens.

When the lens has reached an advanced stage, this mass of branching vessels disappears and little of the tissue mass on the disk from which it springs should be seen at birth.

The closure of the optic cleft at the inferior angle of the disk, along with the atrophic process which removes the tissue just mentioned, is concerned in the development of the physiologic cup.

Various theories have been advanced to explain why this orderly procedure is distorted, and a most interesting group of anomalies resulting from imperfect absorption or construction have been recorded. Some of these have practical importance in the diagnosis of intraocular tumors in infancy and conditions of the retina and disk in later life. Seven of 24 eyes removed for retinoblastoma at the Royal London Ophthalmic Hospital (Moorfields) had embryonic remnants. Congenital detachments in the form of folds require differentiation from traumatic lesions and embedded foreign bodies. Anomalies of the disk do not have as important a place in actual practice, but "pits in the optic disk," abnormal cups and tissue remnants on the disk require explanation.

(Three slides were shown.) The first shows the persistent stem of a hyaloid artery projecting into the vitreous; the next shows an eye with the hyaloid vessels all the way from the disk to the lens capsule. This is a rarity for a single slide. The next slide shows typical pseudoglioma; the vascular capsule is very plain and typical, and the connection with the Bergmeister papilla is clearly demonstrated.

Drusen in the Papilla. DR. BERNARD SAMUELS, New York.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

Book Reviews

Lectures on Motor Anomalies. By Alfred Bielschowsky, Professor of Ophthalmology, Dartmouth Medical School. Price, $1. Pp. 128, with illustrations. Hanover, N. H.: Dartmouth College Publications.

The twelve lectures which comprise this monograph on motor anomalies of the eyes were given before the Research Study Club of Los Angeles in January 1938. In his review of the physiology of ocular movements, Bielschowsky places particular emphasis on deficiency of the fusion faculty as the most important factor in the causation of strabismus and discusses in detail fusional movements of the eyes.

The next two chapters are devoted to consideration of heterophoria, in which the author's well known but debatable views on the importance of anomalous positions of rest as a cause of heterophoria are outlined. A brief but sane discussion of the nonoperative treatment of heterophoria is given.

The various theories on the causation of strabismus are reviewed, with the conclusion that the problem has not been cleared up satisfactorily. The author divides the etiologic factors producing the faulty position of the eyes into three groups: (1) those due to mechanical conditions, (2) those due to excessive or spastic motor innervation and (3) those due to a combination of mechanical and neural factors. He believes, however, that none of these factors produce more than periodic squint as long as the fusion faculty is normal. He furthermore states that the mere absence of the fusion faculty does not cause permanent strabismus. In other words, it is his opinion that a combination of defective fusion with one of the other aforementioned factors is necessary for the development of permanent squint. He then outlines the necessary steps in the examination to determine which of these etiologic factors is the predominant one in any given case. Aniseikonia as cause of defective fusion is mentioned and illustrative cases cited.

In the chapter on principles of surgical treatment the author shows the width of his experience and the soundness of his judgment. He correctly stresses the guiding principles in the selection of operations for strengthening and weakening the muscles of the eye rather than elaborating on the particular technic employed.

The second half of this monograph is devoted to paralyses of the ocular muscles. The symptoms are thoroughly covered in a clearcut manner. The excellent summary of the behavior of diplopia in the different kinds of pareses should prove useful to any student of ophthalmic myology. Several special types of paralyses of the ocular muscles are discussed and are accompanied with photographs of illustrative cases. These chapters are well worth reading, for they contain many interesting comments on some of the more unusual types of ophthalmoplegias. The experience of many ophthalmologists

is not in accord with the author's statement that "by far the most frequent and important type of paralysis of a single vertical motor is trochlear-nerve palsy." A good review of paralyses of the supranuclear type follows, and the causation of ocular paralyses is briefly covered with emphasis on the frequency of palsy of the trochlear nerve following a radical operation on the frontal sinus. In the discussion of surgical treatment of paralysis the author does not favor transplantation of the temporal halves of the superior and inferior rectus muscles in case of paralysis of the external rectus muscle. He correctly calls attention to the fact that in all the successful cases the transplantation is combined with a weakening of the internal rectus muscle. His surgical management of the other types of paralyses is in accord with the generally accepted views. A short chapter on ocular spasm concludes the monograph.

This monograph is to be recommended to the ophthalmologist, for it not only portrays the views of one of the outstanding students in the field of ophthalmic myology but contains many practical suggestions not found in a formal textbook. JOHN H. DUNNINGTON.

Festschrift in Honor of the Sixtieth Birthday of Prof. Dr. Alfred Vogt. Contributed by his Friends and Thankful Pupils. Price, 28 francs (16.80 marks). Pp. 912. Basel: Benno Schwabe & Co., 1939.

After a foreword from the Swiss Ophthalmological Society and from the *Festschrift* Committee, which includes the following names, Drs. M. Bücklers, A. Franceschetti, O. Haab, W. R. Hep, R. Klainguti, K. Rehsteiner, H. Schläpfer, W. F. Schnyder, H. Wagner, A. Wiederkehr and H. Zangger, Drs. Schnyder and Rehsteiner give an interesting description of Professor Vogt's life as an introduction to this *Festschrift*, of which the following is an abstract.

As a student Vogt was devoted to natural sciences. He received his ophthalmic training as assistant to Mellinger and then became director of the department of ophthalmology of the cantonal hospital in Aarau, which under his guidance rapidly grew in importance. His first publication was on the effect of ultraviolet rays on the eye. He gave the first correct interpretation of erythropia. Then followed studies on the ultra-red rays and their effect on the eye, which are important in explaining the cause of the cataract of glassblowers. Vogt showed that the ultra-red short waves are the only ones that can injure the crystalline lens. His next contribution was on ophthalmoscopy in red-free light, which allows the recognition of the nerve fibers in the retina, the retinal blood vessels, the yellow color of the macula, certain retinal reflexes, cyst formations in the retina and fine opacities of the vitreous. These conditions are visible when the reflected red light from the choroidal vessels is dampened. In 1913 Vogt showed that the incipiency of a cataract occurred in the perinuclear zone and not in the subcapsular zone as Hess believed. His contributions on the anatomy and pathology of the lens were fundamental and marked a great advance in the knowledge of this subject. Vogt was called to Basel in 1917 and to Zurich in 1923.

This period was devoted to his remarkable investigations with the slit lamp, and his name will always be connected with the microscopic studies in the living by which the knowledge of corneal and lenticular changes have been so far advanced. Vogt was one of the first after Gonin's discovery to study the new problem of retinal detachment, and he published one of the best textbooks on this subject, "Die operative Behandlung der Netzhautablösung," in 1936, and perfected the operation with perforating cathode electrolysis. A subject in which he was always interested and returned to from time to time is heredity and its important role in certain ophthalmic conditions.

A list of Vogt's publications follows, commencing with the year 1905 and reaching 223 in 1939, with titles of 241 demonstrations and papers presented at medical meetings and a list of 195 articles which have been published by his assistants.

The *Festschrift* contains 97 articles, divided into those which appeared in *Schweizerische medizinische Wochenschrift, Klinische Monatsblätter für Augenheilkunde* and *Archiv für Ophthalmologie.*

This remarkable collection of articles, which covers many fields of ophthalmology and general medicine, is a most fitting memorial to a man whose tremendous working power, keen observation and devotion to science have placed him among the great ophthalmologists.

ARNOLD KNAPP.

Iridosclerectomy with Anterior Cyclodialysis for Glaucoma. Geraldo Queiroga. Thesis presented for doctor's degree to the Faculty of Medicine, University of Minas Geraes, Bello Horizonte, Brazil, June 1939. Pp. 43, with five illustrations.

The author's technic is a combination of sclerectomy, anterior cyclodialysis and peripheral iridectomy. After dissecting a conjunctival flap down to the limbus above, he makes two parallel horizontal incisions partly through the sclera, 1 mm. apart and 2 mm. from the limbus and approximately 3 mm. long. One of the scleral incisions is carried deeper until aqueous appears at one point. The sclera here is then grasped with fixation forceps, and the 1 by 3 mm. piece of sclera is excised with scissors. A spatula, bent almost at right angles, is inserted posteriorly into the filtration angle, through the scleral spur and into the suprachoroidal space and is rotated over a distance of 90 degrees. If peripheral synechiae are present, the spatula is inserted anteriorly and the synechiae freed. A peripheral basal iridectomy is then done.

The operation is indicated in all types of glaucoma except acute glaucoma and secondary glaucoma from intraocular inflammation, from an intraocular new growth and from a subluxated lens. Twenty-one eyes were operated on, and the operation reduced the intraocular tension to normal in every case. Some of the patients were not followed after their discharge from the hospital; others were followed only several weeks, and none was followed longer than several months.

A. B. REESE.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chinese Ophthalmology Society

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Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. **Time:** Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. H. G. Ditroi, Szeged.
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, Maria ucca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.
Place: Perth, Western Australia. Time: Sept. 2 and 7, 1940.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIEDAD OFTALMOLOGIA DEL LITORAL, ROSARIO (ARGENTINA)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Juan M. Vila Ortiz, Córdoba 1433, Rosario.
Place: Rosario. Time: Last Saturday of every month, April to November, inclusive.
All correspondence should be addressed to the President.

SOCIEDADE DE OPHTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Francisco Ferreira, Pitangueiras 15, Brotas, S. Salvador, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

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Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

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Secretary: Dr. K. O. Granström, Södermalmstorg 4 IIIl tr., Stockholm, Sö.

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President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Albert C. Snell, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary: Dr. Derrick Vail, 441 Vine St., Cincinnati.
Place: Cleveland. Time: June 2-6, 1941.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

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Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

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President: Dr. F. P. Calhoun, 478 Peachtree St. N. E., Atlanta, Ga.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

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Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. H. Lowry, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. Alexander E. MacDonald, 421 Medical Arts Bldg., Toronto.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

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Executive Director: Mrs. Eleanor Brown Merrill, 50 W. 50th St., New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Charles W. Barkhorn, 223 Roseville Ave., Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. V. May, 1703 Main St., Marinette.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Wisconsin Rapids. Time: June 1941.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. William D. Rowland, 84 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Isaac H. Jones, 635 S. Westlake Ave., Los Angeles, Calif.
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President: Dr. Clarence W. Shannon, 4th and Pike Bldg., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1215–4th Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

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Secretary-Treasurer: Dr. J. J. Potter, 303 N. Main St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

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Secretary-Treasurer: Dr. J. H. Curts, 330 S. Washington Ave., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

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Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

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Secretary: Dr. A. E. Cruthirds, 15 E. Monroe St., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. J. K. Heckert, 103 N. Washington St., Lansing.
Secretary-Treasurer: Dr. W. D. Irwin, 710 Hanselman Bldg., Kalamazoo.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. H. Bailey, 29 S. Oakland Ave., Sharon.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

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Secretary-Treasurer: Dr. Raymond C. Cook, 701 Main St., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. William M. Bane, 1612 Tremont Pl., Denver.
Secretary: Dr. Harry Shankel, Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. W. E. McClellan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. E. N. Maner, 247 Bull St., Savannah.
Secretary-Treasurer: Dr. C. K. McLaughlin, 567 Walnut St., Macon.
Place: Macon. Time: May 13-17, 1941.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

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Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

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Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Cedar Rapids. Time: November 1940.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Henry N. Blum, 912 American Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

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Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Erling Wilhelm Hansen, 78 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

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Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

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Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. Edgar P. Cardwell, 47 Central Ave., Newark.
Secretary: Dr. Arthur E. Sherman, 243 S. Harrison St., East Orange.
Place: Atlantic City. Time: June 3-5, 1941.

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Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

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Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Secretary-Treasurer: Dr. R. S. Fixott, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. W. Carpenter, 200 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia. Time: Nov. 5, 1940.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. J. W. Ward, 2607½ Lee St., Greenville.
Secretary: Dr. Dan Brannin, 929 Medical Arts Bldg., Dallas.
Place: Fort Worth. Time: Dec. 13-14, 1940.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. Rowland H. Merrill, 1010 First National Bank Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Earl H. Phillips, 628 Judge Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. George G. Hawkins, Newport News.
Secretary-Treasurer: Dr. Guy Fisher, 3 E. Beverley St., Staunton.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George E. Black, 7 W. Bowery St., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Stacy C. Howell, 144 Ponce de Leon Ave. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Pliace: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Maurice Wieselthier, 1322 Union St., Brooklyn.
Secretary-Treasurer: Dr. Harold F. Schilback, 142 Joralemon St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. James G. Fowler, 412 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Richard Gamble, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Vernon M. Leech, 55 E. Washington St., Chicago.
Place: Chicago Towers Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmologic Club

Chairman: Dr. Albert D. Ruedemann, Cleveland Clinic, Cleveland.
Secretary: Dr. B. J. Wolpaw, 2323 Prospect Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Francis H. Adler, 313 S. 17th St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. D. Postle, 240 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. F. K. Stroud, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. Arthur Padilla, 414 Medical Professional Bldg., Corpus Christi, Texas.
Time: Second Friday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Dan Brannin, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. L. E. Darrough, 4105 Live Oak St., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Detroit Ophthalmological Society

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. A. E. Jackson, 602 W. 10th St., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. Charles Dickson, 1617 Medical Arts Bldg., Houston, Texas.
Secretary: Dr. William J. Snow, 708 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. M. Masters, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Homer Beal, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Desmond Curran, Federal Reserve Bank Bldg., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Paul Southgate, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Kirt G. Parks, 605 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. Colby Hall, 1136 W. 6th St., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE AND EAR SOCIETY

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. E. J. Cummings, 1835 I St. N. W., Washington.
Secretary: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Secretary-Treasurer: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Secretary: Dr. Guy Maness, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW HAVEN OPHTHALMOLOGICAL SOCIETY

President: Dr. Leonard C. Whiting, 121 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Algernon Reese, 73 E. 71st St., New York.
Secretary: Dr. Brittain Payne, 35 E. 70th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President Dr. Morris Davidson, 80 Centre St., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Leatherock, Cushing, Okla.
Secretary: Dr. Harry C. Ford, 1014 Medical Arts Bldg., Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. Howard Heine, 635 N. Main St., Fremont, Neb.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. L. Markowitz, 16 Church St., Paterson, N. J.
Secretary-Treasurer: Dr. A. John Reinhorn, 302 Broadway, Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward A. Weisser, 119–5th Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. F. Bryce, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

St. Louis Ophthalmic Society

President: Dr. J. F. Hardesty, Missouri Theatre Bldg., St. Louis.
Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Dan Russell, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. P. G. Bowen, 315 Camden St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Matthew Hosmer, 384 Post St., San Francisco.
Secretary: Dr. Fred Boyle, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Louis A. Parsell, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. F. R. Webster, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. John R. Myers, State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. S. H. Patterson, 1251–15th St., Toledo, Ohio.
Secretary: Dr. J. E. Minns, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Frank Costenbader, 1726 I St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. L. Conner Moss, 1710 Rhode Island Ave. N.W., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.